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The Journal of Bone and Joint Surgery

EDUCATION AND CERTIFICATION OF ORTHOPAEDIC SURGEONS IN THE UNITED STATES *

BY J. ALBERT KEY, M.D., ST. LOUIS, MISSOURI

A considerable number of the physicians who are now being separated from the Army have become interested in orthopaedic surgery and wish to acquire more knowledge of the subject and to practise this specialty in civil life. There is also an increasing number of young medical graduates who choose to enter this field. It thus seems appropriate to consider what course of study, training, and experience should be pursued in order to qualify a doctor of medicine as an orthopaedic surgeon.

During the past twelve years considerable time and thought have been devoted to the subject, and an answer has been arrived at which is satisfactory to many orthopaedic surgeons. This is indicated by the fact that The American Academy of Orthopaedic Surgeons and the Central States Orthopaedic Society require certification for membership. The American Orthopaedic Association can waive certification by action of the Executive Committee, but I believe that only one member who was not certified has been elected to our Association during the past twelve years.

Not only does admission to each of the three large orthopaedic societies in this country depend upon certification, but many hospitals are now requiring certification by a national board before a surgeon or physician can practise a surgical or medical specialty in the hospital. Likewise, some government agencies including the Veterans Administration are veering in the same direction. It is thus evident that no one should at present even consider entering the field of orthopaedic surgery in this country, unless he is in a position to fulfill the requirements for examination with the expectation that, if he passes the examination, he will then be certified as qualified to practise orthopaedic surgery.

We now have American boards for the examination and certification of practitioners in practically every major medical and surgical specialty. The fundamental reason for the existence of each of these boards is to establish minimum standards for the practice of the specialty of that board. I am told that the operation of these boards has greatly improved the quality of the work done in the various medical specialties in our country. I wonder if this is true. It is possible that the ardent advocates of specialty boards are taking credit for changes and improvements which result as the natural development of medical science, in the same way that an advocate of a given therapeutic measure cannot resist crediting his treatment with the cure of a self-limited disease. However, the practice of medicine in this country was not very sick. Our specialists have been doing good work

* Presidential Address presented at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 28, 1946.

for a long time. Consequently, our boards may be taking credit for curing a patient who was in good health when the treatment was started. And this is especially true of orthopaedic surgery. On the other hand, it may be accepted that the boards, by approving certain institutions for resident training in particular specialties, have improved or at least caused the expansion of graduate training in the various specialties. Whether or not this increasing emphasis on specialization in the practice of medicine is desirable is beyond the scope of this paper.

The American Board of Orthopaedic Surgery has made the condition that, in order for a man to qualify for the examination leading to certification, he must have completed the following courses of training in accredited institutions:

1. One year rotating internship;
2. One year internship in adult orthopaedic surgery;
3. One year internship in children's orthopaedic surgery;
4. One-half year internship in fractures;
5. One-half year full-time study of fundamental subjects (anatomy, pathology, bacteriology, physiology, and biochemistry);
6. Two years in practice limited to orthopaedic surgery.

No one will deny that the above is very satisfactory as a minimum standard for the training of an orthopaedic surgeon; but, too, I do not think that any one will be willing to defend the thesis that the completion of the above course of training is the *only* method by which a physician can become a qualified orthopaedic surgeon.

Due to the unswerving devotion to duty of the Membership Committees, our American Orthopaedic Association comprises a group of orthopaedic surgeons whose ability cannot be questioned; and yet I doubt if 10 per cent. of our present membership could meet the present training requirements. It is thus evident that it must be possible for one to become proficient in orthopaedic surgery by other routes.

For many years I have been interested in the careers of our members, and in why and how certain of them became outstanding in their field. I still do not know the answer. Each man has pursued a different course, and there are many reasons to which the success of these individuals can be attributed. Part of it is of course due to natural ability and intelligence. If you wish to make a silk purse, you must have some silk to start with. Part of it is due to an unusual capacity and desire on the part of the individual to work hard and long over a period of years. Part of it is due to luck, and this includes a favorable environment and opportunities which were seized when they were presented, as well as occasional fortuitous events or discoveries. Part of it is due to education, and a surprisingly large part of this education consists, not of formal training, but of informal association with men of talent and learning and of self-directed study. Most successful men in medicine continue to study and to learn as long as they practise, and their stature continues to increase until they reach the stage where they are forgetting more than they learn.

It would seem then that a scheme of education which would produce a group of orthopaedic surgeons comparable to that comprising our membership would be a very satisfactory answer to our problem. However, it is evident from a study of the education of our members that they did not pursue a uniform course of training. Our membership varies widely in general education, in scientific ability, in surgical skill, in knowledge of disease, and in erudition; and yet each of these men has certain qualities which make him worthy of membership.

I wonder if we are not making a grave mistake in our attempt to regiment the training of orthopaedic surgery. To limit future orthopaedic surgeons to those of our young medical graduates who can be forced into a mold is to exclude many whose qualifications justly entitle them to certification and whose abilities as orthopaedic surgeons would be of great value.

Certainly the present system will produce a group of uniformly well-trained orthopaedic surgeons of moderate ability, and some of more than average ability will emerge from the assembly line. On the other hand, orthopaedic surgery will suffer because of this uniformity, and many men will have been forced to submit to courses of training which were unsuitable answers to their problems; while others who might have become outstanding orthopaedic surgeons will be denied the privilege of entering this specialty, because they did not decide to adopt it before or shortly after finishing medical school. They properly waited until their judgment was more mature. By this time they had developed skill in general surgery or general medicine for which they could get no credit from our Board, or they had incurred obligations which prevented them from starting all over again and devoting three full years to so-called formal specialized training.

From what has been said, one may infer that I believe that our present program may actually be hindering, rather than contributing to, the advance of our specialty. From time to time we should examine critically our requirements for certification, in order to determine whether or not our plan is functioning in a manner which is satisfactory to us. I submit the following:

1. We should study modern methods of examinations, especially those developed in the Army and, if necessary, should seek the aid of psychologists. We can devise comprehensive examinations which will be real tests of a man's ability. With such examinations it would be possible to be less rigid in the requirements for preliminary training. It is to be emphasized that we owe more to the candidates than they owe us. In planning the examinations, it should be recognized that the orthopaedic surgeon is a surgeon who largely limits his work to the spine and extremities, and it is the function of the Board to determine whether or not the candidate's knowledge and ability are such that he can be classified as an expert in this field. His qualification to practise medicine has already been determined by his state or the national board of medical examiners. Uniform examinations in anatomy, physiology, pathology, biochemistry, and bacteriology, to be taken by the candidates for certification in general surgery and in all of the surgical specialties, are not suitable for the purpose intended.

Such examinations would be planned largely to test the candidate's knowledge of general surgery, and would not test his proficiency in orthopaedic surgery. While the term general surgeon may be taken as referring to a general practitioner of surgery, who is justified in performing any type of surgical procedure, it is a fact that in areas in which the various surgical specialties are adequately covered, the general surgeon largely limits his practice to the abdomen, breast, front of the neck, and the subcutaneous tissues. It is thus evident that his field is more restricted than is that of the orthopaedic surgeon. We must abandon the delusion that the modern general surgeon is proficient in all of the surgical specialties. It is obvious that each surgical specialty should conduct its own examinations and is capable of determining whether or not a man is qualified to practise that surgical specialty.

2. We should revert to our original function of establishing minimum requirements for the practice of orthopaedic surgery and should avoid even the appearance of attempting to dictate the future development of our specialty. With this in view we should abolish the requirements of certification for membership in our orthopaedic societies. We should also discourage hospitals and government agencies from discriminating for or against an orthopaedic surgeon because he has or has not been certified by the Board.

We may lessen the number of candidates for examination, but we will improve the quality and will tend to eliminate the rapidly increasing throng of half-baked young orthopaedic surgeons whose only claim to fame is that they are "certified", and who, having attained that enviable position, can look down on their less fortunate brethren and from now on can rest on the laurels gained by six years spent in pursuing the course prescribed. If left to follow their own inclinations in their desire to perfect themselves in

orthopaedic surgery, how many of these men would have pursued the same or a similar course? Is it not likely that some of the more adventurous spirits would have deliberately or casually left the beaten path for a while and wandered into the territory allotted to allied branches of medicine or related sciences? And when they returned, laden with the fruits of their labors in other fields, would this increment be harmful to orthopaedic surgery? Our specialty needs all of the outside help that it can get, and we should welcome men who are learned in other branches of medicine. As we narrow and lengthen the road, we progressively lessen the expansion of our specialty and its ability to serve mankind.

3. This brings me to a subject where there is room for wide divergence of opinion,—namely, the minimum requirements for training before a candidate can be accepted for examination leading to certification. It is my impression that we have been too rigid in our insistence that candidates fulfill the requirements as regards preliminary training. We should demand evidence that the candidate has devoted sufficient time to the study and practice of orthopaedic surgery, so that he may reasonably be expected to have acquired a satisfactory knowledge of the subject and sufficient skill in the technique of orthopaedic procedures to enable him to practise his specialty with safety, and to have developed adequate maturity of surgical judgment and scientific integrity to warrant his being entrusted with the orthopaedic problems arising in his community. The candidate should then be subjected to a searching examination, in order to determine whether or not he has profited sufficiently from his study and experience to merit the stamp of approval.

We should cultivate broadness of mind in giving credit for the work done in other branches of medicine or surgery or of allied sciences, and in this way attract to our specialty men who have something more to contribute than their knowledge of orthopaedic surgery. Likewise, they should not hesitate to give ample credit for years of experience in the practice and teaching of orthopaedic surgery in recognized clinics or hospitals or in association with orthopaedic surgeons of known ability. The function of the Board is to determine whether or not the candidate possesses the minimum requirements for an orthopaedic surgeon, and it should not be too greatly concerned about how this ability was acquired. We should eliminate the requirement that a candidate rigidly apportion his time among adult and children's orthopaedics and fractures, and especially should we abandon the required six months spent in reviewing the fundamental branches of medicine, presumably in their relation to orthopaedic surgery. I know of no place where such a course can be obtained and, if it is ever offered in a medical school, it will be a super-quiz course; and those who take it will have wasted time in acquiring a mass of ephemeral knowledge, which could have been spent more profitably in research or in clinical work.

The candidate should know that he will be examined in the anatomy and pathology of his specialty and that he may be asked questions involving knowledge of other fundamental sciences; and he will attempt to increase his knowledge of them as he pursues his orthopaedic education. To demand that an orthopaedic surgeon forsake his practice and spend six months cramming fundamental sciences into his brain is certainly verging on the ridiculous.

I am convinced that regimentation of postgraduate medical education in the specialties is a mistake. No one specific course of study or hospital training is suitable or desirable for all men who are to engage in the practice of orthopaedic surgery. If I were to attempt to lay out a course for a hundred such men, I would start in high school and see that, in addition to their regular studies, all were given a fairly comprehensive course in manual training, in order that they might learn to use their hands and acquire knowledge of and respect for sharp tools.

All would enter college and about half of them would continue until graduation. These would take a variety of courses: some, science; some, engineering; some, the classics; some, humanities; and some, business administration with enough science to meet the entrance requirements of the medical school of their choice. An effort would be made

to stimulate each student to develop especial interest in one or two subjects, perhaps not related to medicine, during the last two years of college. About a fourth of these would spend a year in graduate study before entering medical school, another fourth would work for a year or so, and the others would enter at the next term after graduation from college.

The other half, largely because of limited financial resources, would spend only two or three years in college. Their object would be to fulfill the entrance requirements to medical school and to acquire as adequate a general education as possible within the time allotted. This heterogeneous group would then complete the regular course leading to the degree of Doctor of Medicine in a Class A medical school, and each of them would then serve a one-year internship in surgery; about half of these would then begin their orthopaedic training and would serve for one or more years in an orthopaedic service where the visiting staff were conscious of their obligation to train the resident staff.

Of the other fifty who had completed one year in general surgery, about half would continue in this field for from one to four years longer before beginning their orthopaedic training. The other twenty-five would interrupt their surgical training at this point and spend a year or more in study and research in general medicine or preferably in one of the fundamental branches of medicine. The members of this group would then begin their orthopaedic training two or more years after graduation, and they too would serve one or more years in a satisfactory orthopaedic service.

We now would have a group of young men, all of whom have had one-year internships in general surgery and one-year internships in orthopaedic surgery, and half or more of whom have spent from one to five years additional in training in general surgery or in work in some other branch of medicine. It would be recommended that all of these men spend a second or possibly a third year in an orthopaedic service where they could be given more responsibility and gain more experience, but this would not be required. Some of them would enter practice in association with more mature orthopaedic surgeons; some would enter practice directly and continue to add to their knowledge by study and attendance at medical meetings; some would take full or part-time positions and continue to work in out-patient clinics, and gradually would be entrusted with the care of in-patients and with teaching in established orthopaedic centers.

In this manner, we would obtain a hundred orthopaedic surgeons with wide variation in general and special education and a group which would compare favorably with our present membership.

I would also not fail to recognize the fact that a good general surgeon can develop into a good orthopaedic surgeon by simply directing his energies to that field. Likewise, a young man with training only in general surgery may, by study and association with a good orthopaedic surgeon, become highly skilled in this field. In neither case is any formal orthopaedic training necessary. Would I perhaps be going a bit too far if I even suggested that it is possible for one to practise general surgery and yet be an excellent orthopaedic surgeon, and to voice the opinion that certain general surgeons merit qualification as orthopaedic surgeons, just as some orthopaedic surgeons are excellent general surgeons?

In view of the foregoing, I suggest that we alter radically our policy of demanding that a candidate follow a rigidly outlined course of orthopaedic training, and that we recognize the incontrovertible fact that satisfactory orthopaedic surgeons have been and can be developed in many different ways. Our standards of excellence should not be lowered, and we can demand more time devoted to practice; however, we should not close the door of our specialty in the face of men who are well qualified, but whose education has not been along the lines of regimentation.

SURVIVAL IN BONE SARCOMA*

BY HARRY PLATT, M.S., F.R.C.S., F.A.C.S., MANCHESTER, ENGLAND

Professor of Orthopaedic Surgery, University of Manchester

Most of our general and statistical knowledge regarding prognosis in the various types of bone sarcoma is derived (1) from figures published by the Registry of the American College of Surgeons between the years 1934 and 1939, inclusive; and (2) from such individual contributions as those of W. B. Coley (1933 and 1934), Campbell (1935), B. L. Coley (1938), Meyerding (1938), Simmons (1939), and more recently Macdonald and Budd (1943). Analyses of the not inconsiderable number of cases classified as "long survivals" (patients living five years or more), recorded by the Registry or in the personal studies of the writers already mentioned, permit us to draw certain broad conclusions concerning the effectiveness of various methods of treatment.

The case for the surgical eradication of accessible tumors as the treatment of choice is in my judgment fully proven. The value of postoperative treatment by means of toxins or irradiation or both seems to be still *sub judice*, but these therapeutic agents should not be withheld from a patient if circumstances permit their systematic use.

It can now be said that the outlook in the case of *osteogenic sarcoma* is not so gloomy as it appeared at one time. Of the various histological types, the *spindle-cell sarcoma* (fibrosarcoma) would appear to be the least malignant form, but the evidence regarding the degree of malignancy of the *chondromyxosarcoma* is conflicting. The *extraperiosteal sarcomata*, in my experience, are also tumors of varying malignancy. Of the thirty-two cases in my own collection, six appear in the group of five-year survivals, and seven in the list of short survivals (under two years). All authorities are agreed that the outlook in the *Ewing* tumor remains grave, despite its ready response to irradiation. In this connection, it is important to note that thirteen of the fourteen five-year survivors with this tumor, recorded in the Registry report of 1939, had been treated by surgical measures. These general facts must determine the advice we offer to our patients. But the problem of survival in bone sarcoma still remains an enigma, for so far there has been a failure to elicit any significant evidence concerning the influence of various factors which might possibly have a causal relation to this phenomenon.

I have recently submitted my own modest collection of 161 sarcomata to a scrutiny on the lines laid down by Bradley Coley and Pool in their most valuable paper, published in 1940. An analysis of my series reveals twenty-three five-year survivals. Twenty-two of these patients, treated by radical operations, are fully proven cases, but in one—a pelvic sarcoma—proof from biopsy was not forthcoming.

FIG. 1-A

The main details are presented in Table I.

Among these twenty-three cases were two patients with pelvic sarcomata treated by hindquarter amputation; the survival period for one was five years, and the other is still

* From a paper read before the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 29, 1946.

FIG. 1-C

FIG. 1-B

T. B., male, aged thirty-six years, had osteogenic sarcoma (chondrosarcoma) of the left ilium.
Fig. 1-A: Shows the large sloughing area, following treatment by irradiation for two years.
Fig. 1-B: Roentgenogram shows condition of the ilium at this time (November 9, 1938). Hindquarter amputation was performed in December 1938. The man is alive and well, seven years later.
Fig. 1-C: Drawing of the specimen obtained at operation.

living (in June 1946) after seven years (Figs. 1-A, 1-B, and 1-C). Eight patients in this series survived more than ten years,—five of these had osteogenic sarcomata; two, extraperiosteal sarcomata; and one, a sarcoma arising in a chondroma (Figs. 2-A and 2-B).

I have attempted, without success, to set this series of twenty-three long survivals over against a series of short survivals (under two years) of which my collection affords forty-nine examples, in the hope of discovering a clue to the mystery of long survival. In this comparison, I have found that no significance can be accorded to such factors as age, sex, site of tumor, and the duration of symptoms before the tumor was eradicated, the latter being a point notoriously difficult to estimate accurately. (See Figures 3-A, 3-B, 4-A, and 4-B.) Forty-one patients in this series of short survivals died within a year after operative treatment. At first sight this is a melancholic picture, but when we consider that, in 128 accessible tumors treated by radical operative procedures, seventy-nine patients have survived over two years, and twenty-three of these five years or more, the current view, that the general outlook in bone sarcomata as a whole is by no means tragic, is reinforced.

I have been unable to use the Coley toxins systematically, either following operation or in inoperable tumors, so that I have had no opportunity of estimating the part they may play in determining survival.

I believe that in the combination of radical operation and some systemic agent, still to be discovered, lies the hope of potential cure or still longer survival in this field of malignant disease. Irradiation is admittedly a useless form of therapy in the tumors of

FIG. 2-A

FIG. 2-B

R. C., a boy, eleven years old, had osteogenic sarcoma of the upper end of the left tibia.
Fig. 2-A: Shows condition of knee before operation.
Fig. 2-B: Roentgenogram showing large osteolytic tumor involving the metaphysis of the upper end of the tibia. Amputation was performed January 13, 1936, and a regular course of Coley's toxins was given during the three years immediately following amputation. The boy is living and well, ten years after operation.

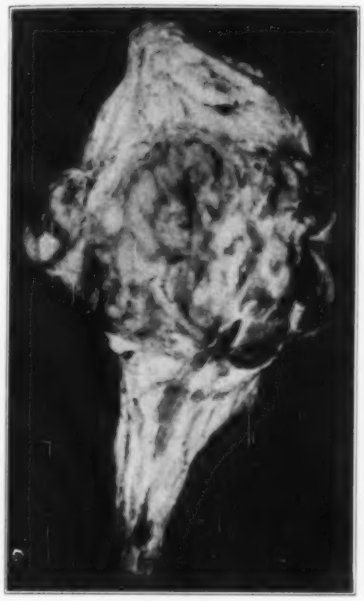

FIG. 3-A

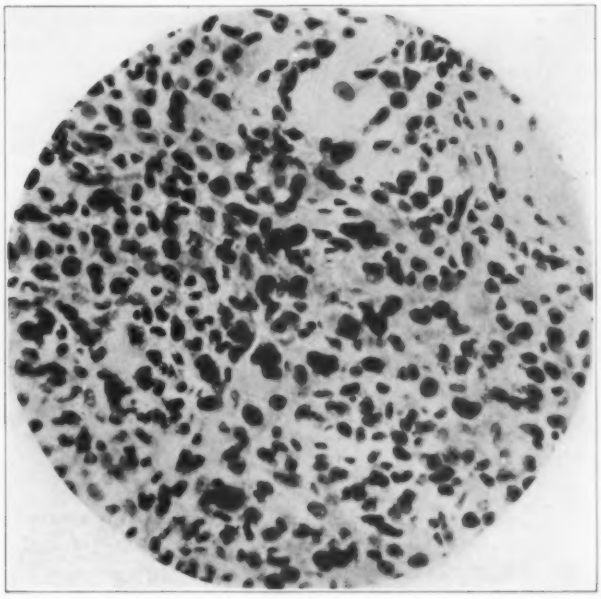

FIG. 3-B

H. R., male, aged twenty-three years, had osteogenic sarcoma of the upper end of the left fibula.
Fig. 3-A: Shows amputated specimen.
Fig. 3-B: Photomicrograph showing that the predominant cell was oat-shaped. The man died eighteen months after amputation.

FIG. 4-A

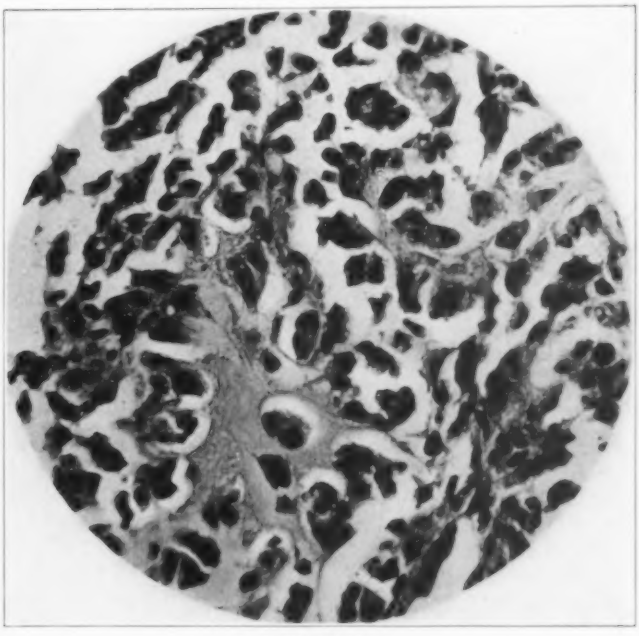

FIG. 4-B

K. P., male, aged twenty-four years, had osteogenic sarcoma of the upper end of the right fibula.
Fig. 4-A: Shows amputated specimen (compare with Fig. 3-A).
Fig. 4-B: Photomicrograph shows that the predominant cell was oat-shaped. Patient is living, thirteen years after amputation.

the osteogenic-sarcoma group. My own slender experience with the Ewing tumor has not convinced me that irradiation before amputation, or irradiation alone in accessible tumors, has any material influence on the ultimate fate of the victims of this most lethal type of bone tumor.

TABLE I

LONG SURVIVALS (5 YEARS AND OVER)

Type of Lesion	No. of Cases	Totals
Osteogenic sarcoma		
Sclerosing	0	
Osteolytic	5	
Chondromyxosarcoma	8	13
Sarcoma in abnormal bones		
Fibrocystic disease	1	
Benign osteochondroma	3	
Paget's disease	0	4
Extraperiosteal sarcoma		6
Ewing's tumor		0

Age of Patient at First Treatment	No. of Cases	Survival Periods	No. of Cases
Under 20	4	5 to 9 years	15
20 to 40	12	10 to 15 years	6
40 and over	7	16 to 20 years	2

TABLE II

SHORT SURVIVALS (UNDER 2 YEARS)

Type of Lesion	No. of Cases	Totals
Osteogenic sarcoma		
Sclerosing	4	
Osteolytic	23	
Chondromyxosarcoma	6	33
Sarcoma in abnormal bones		
Paget's disease	4	
Osteochondroma	1	5
Extraperiosteal sarcoma		7
Ewing's tumor		4

CONCLUSIONS

A study of a series of twenty-three five-year survivals when compared with a series of forty-nine short survivals (under two years) fails to reveal any outstanding or significant factors which would determine the prognosis in the sarcomata of bone.

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DISCUSSION

DR. D. B. PHEMISTER, CHICAGO, ILLINOIS: It has been most stimulating and interesting to hear this presentation of Professor Platt's on bone sarcoma, based on his careful study of a large series of cases.

He spoke of the appearance of bone sarcoma in bones that have been the sites of previous changes. The occurrence of sarcoma in bone that has been irritated might be mentioned. At the last meeting of this Association, Dr. C. Howard Hatcher reported three such cases. Because of the known appearance of sarcoma in the skeleton after radium ingestion, it is certain that uranium and plutonium ingestion will predispose to the same development.

About benign giant-cell tumors, I think we are quite generally agreed in this country that surgery is the best form of treatment. Personally, I feel that amputation should rarely be done, but instead the more extreme cases should be treated by resection and bone transplantation.

As to the classification of bone sarcomata, I feel that in some cases one should be satisfied with the diagnosis of sarcoma and not be too much concerned about the type of cell or tissue. However, a knowledge of the cell or tissue type is desirable, since it may aid in the selection of the best form of treatment. In recent years, the use of large blood transfusions has made it possible to do very extensive resection of bone and soft parts at the tumor site and to repair the skeletal defect by massive bone transplantation, without risk of circulatory failure; and penicillin cuts down the risk of infection and loss of the bone graft. This makes excision possible in certain favorable cases and avoids amputation. From the standpoint of classification, the most favorable types of sarcoma for resection, in our small series of cases, have been chondrosarcoma and sarcoma arising in giant-cell tumor of bone. Four chondrosarcomata have been excised and replaced by bone grafts, and the patients have survived from six to twelve years without regional recurrence or metastases. Likewise, two patients with sarcomata developing in benign giant-cell tumors have remained well, one for five and one-half, and one for eleven years following excision. Osteogenic sarcomata, regardless of how small they were at the time of operation, have shown a marked tendency to make distant metastases and to recur locally. However, in one classical case of sarcoma of the upper humerus which was ossifying markedly, the patient is well, with a useful arm, ten years after resection and transplantation. One patient with a Ewing's sarcoma of the femur, treated first by x-ray and then by resection of seventeen centimeters of the shaft, is alive and free from recurrence six and one-fourth years after operation.

DR. PHILIP D. WILSON, NEW YORK, N. Y.: It is a great pleasure to be allowed to say something in the discussion of Mr. Harry Platt's paper. He is an old friend and one whom I have seen work and one whose work I value very highly. I have been very interested in his remarks, which I think are very informative and stimulating. I think his first point on the responsibility of the orthopaedic surgeon in the care of these cases is a very important one. I am very happy I can make this remark with Dr. D. B. Phemister, who is a professor both of surgery and of orthopaedic surgery, preceding me in the discussion. I know in several centers there are excellent general surgeons caring for these patients and doing excellent work; yet I know of one instance not long ago where a Brodie's abscess was explored under the diagnosis of neoplasm. It needed exploration; it was the proper thing to do; but it seems to me a knowledge of bone lesions or bone pathology is necessary and certainly will help in the diagnosis. I think these men who are treating tumors will invariably have brought to their clinics a great many cases of non-neoplastic conditions, which represent bone anomalies of various types which require orthopaedic care. They will undertake their treatment, when they are not properly qualified for it. I am sure there are many of these lesions where local excision could be done, if the surgeon had a knowledge of the proper reconstruction of bone.

I am very interested in Professor Platt's classification, which is simple and desirable. I would like to tease him a little bit by asking him where he has put osteoid osteoma. I have had the experience of finding it may still be classified as a bone tumor. Again, he excludes from his classification eosinophilic granuloma which, of course, is a questionable diagnosis. We have the feeling that that is a part of a more generalized disease, involving the lipoid and cholesterol metabolism; but I would like to hear Professor Platt's comment on that lesion.

In cases of abnormal bone, I am inclined to feel that the percentage given of five to ten per cent. for the development of osteogenic sarcoma is high. I am sure we are seeing a great many more cases of Paget's disease than formerly; this is largely due to better diagnosis. The localized forms, in which Paget's disease may be confined to a single bone or one or two bones, as well as the generalized cases, are met with. I have had the experience of seeing osteogenic sarcoma in only two cases of Paget's disease, and both have been rapidly fatal. Another condition in which we may see the development of osteogenic sarcoma is chondrodystrophy. I have such a patient, who has survived ten years following excision of the upper end of the femur. I had the unfortunate experience of having to take care of a girl with radium poisoning from emanations in the expired air, in whom osteogenic sarcoma developed. I would like to ask Professor Platt whether he has had experience or whether he cares to make any comment on the A.C.S. serum of the Russians.

Dr. Henry W. Meyerding, Rochester, Minnesota: Professor Platt, it has been a great pleasure to be here and take part in the discussion of your paper. We, as surgeons, are interested in the distinction between benign and malignant lesions of bone, and it is good to see that you have microscopic proof to verify your findings. In 1938 I read a paper on osteogenic sarcoma before the American Medical Association, in which the degree of malignancy of this lesion was graded; I had hoped that you had graded the degree of malignancy in your cases, for knowledge of the grade is of vital importance in deciding the type of treatment to be used. In our series of cases, the patient who had a Grade 1 osteogenic sarcoma did not undergo amputation; whereas among those patients who had Grade 3 or Grade 4 osteogenic sarcomata, the percentage of amputations was high.

The responsibility of early diagnosis rests primarily with the physician who examines the patient first. Later, when the surgeon is called in consultation, he must first exclude pulmonary metastasis, by means of roentgenographic examination of the thorax. We make roentgenograms of the thorax routinely in our cases of tumors of bone. Time and time again I have seen a young patient with a tumor that was not very large; yet I have been amazed by the fact that metastasis to the lungs had already developed. That occurred twice in one day this summer.

Having obtained the history, roentgenographic and laboratory observations, the physician nevertheless may not be able to determine the character of the lesion until he has had resort to biopsy. I am pleased to learn that Professor Platt believes in biopsy. I do not believe in needle biopsy, but prefer that which includes incision, inspection of the tumor, and removal of an adequate portion of tissue for pathological examination. I believe, furthermore, that even though microscopic section and examination and roentgenographic examination are employed, the true character of the lesion may not be known until metastasis or death has occurred. This is a well-known fact. A careful follow-up record in the study of these cases of tumors of bone is very important. These studies are very important; for instance, this summer a patient whom I had first seen many years previously came in to see me. Twenty-six years ago, I had performed for this patient amputation of a leg, through the lower part of the femur, because of a malignant tumor. This man never had answered any of my inquiries concerning his state of health, and I had decided that he was not living. He came back because he had become alarmed at the possibility of recurrence; but we were most pleased to find that his fears were groundless.

In closing, Professor Platt, I will say again that it is a very great pleasure to have you with us, and I appreciate the opportunity of taking part in the discussion of your paper.

Mr. Harry Platt, Manchester, England (closing): About Dr. Phemister's and Dr. Wilson's remarks that metastases preclude the carrying out of resection rather than amputation, I have had little experience with this heroic attack, but I feel now that this line of attack is wiser.

About the eosinophilic tumor that belongs to bone-marrow disease, I do not know.

I do not know anything about the Russian serum. I have heard of it, but I do not know it.

Dr. Meyerding spoke about the grading of malignant tumors, which for many years, in other tumors, has been one of the great achievements of the Pathology Department of the Mayo Clinic. We have not followed that analysis at all, so I cannot say very much about it. I am glad he is a wholehearted supporter of real biopsy and real exposure and removal of tissue. I wish to emphasize once again that I think these tumors, benign and malignant, belong to the skeletal tissues and are, therefore, part of the essential field of modern orthopaedics. Their careful documentation in various clinics is all-important. It is just as important as, if not more important than, study made in an impersonal way on material collected in the literature.

COMPLICATIONS OF FRACTURES OF THE NECK OF THE FEMUR *

BY H. B. BOYD, M.D., AND I. L. GEORGE, M.D., MEMPHIS, TENNESSEE

From the Willis C. Campbell Clinic, Memphis

It is the purpose of this paper to present the complications and end results of internal fixation of 300 acute central fractures of the neck of the femur treated by the Staff of the Campbell Clinic. Smith-Petersen nails were used in 285 of the hips and Knowles pins were used in fifteen. All impacted fractures and fractures of the base of the neck were excluded, only Pauwel[1,2] type II and III fractures being included. In all cases, one year or longer has elapsed since operation.

This is not a consecutive series; none of the patients treated at John Gaston (City) Hospital were included, and most of those treated in the other hospitals in the city were not included, for the reason that roentgenograms on file at the Clinic were inadequate. From a practical standpoint, however, the series may be considered a consecutive one, as the cases were unselected and were consecutive with respect to available and adequate clinical and roentgenographic data. The study includes the early cases on record at the Clinic and was arbitrarily limited to 300 hips, that statistics might be based on an even number.

These 300 fractures of the femoral neck were observed in 294 patients. In none of the six bilateral fractures were both hips injured simultaneously; the shortest period between fractures of the two hips was one year and eight months, and the longest two years and nine months.

TABLE I

MORTALITY

Age	Number of Hips	Deaths	
		Number	Per Cent.
Under 40	15	0	0
40–59	50	0	0
60–69	92	5	5.4
70–79	87	9	10.3
80–96	56	14	25.0
Total	300	28	9.3

Of the total number of deaths, twenty-one occurred in less than four weeks after operation, and seven, within four weeks to six months after operation.

The mortality for the various age groups is indicated in Table I, the overall mortality being 9.3 per cent. There were twenty-eight deaths following the 300 operations, twenty-one of these having occurred within the first four weeks. This is believed to represent the true operative mortality. Seven deaths were recorded from four weeks to six months after operation. Undoubtedly, a number of other patients, whom we were unable to trace, died within the period of four weeks to six months after operation. It is of interest that all of the patients who died were over sixty years of age, and that the mortality increased with age until it reached 25 per cent. in the ninth and tenth decades.

The postoperative follow-up of 131 of the fractures was less than one year (Table II). Over 80 per cent. of the patients treated at the Clinic came from out of the city, and several factors—such as advanced age, poor general health, insufficient funds, or difficulty in se-

* Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 29, 1946.

TABLE II

FOLLOW-UP STUDY

Period	Hips	
	Number	Per Cent.
Less than 1 year	131	43.7
1–2 years	98	32.7
Over 2 years	43	14.3
Deaths	28	9.3
Totals	300	100.0

curing transportation during gas-rationing—prevented follow-up visits. An additional 141 were followed for one year or longer. Bony union took place in 86.5 per cent. of the hips, the bone failing to unite in the remaining 13.5 per cent. (Table III). The majority of patients should be followed for at least a year to determine with certainty whether or not bony union has occurred. In patients of the younger age groups, definite bony union may be seen in the roentgenograms within less than a year. Frequently, however, mistakes will be made if one endeavors to determine the presence of union under one year. Occasionally, bony union develops after a year, especially in patients with gradual absorption of the neck of the femur. In ten of the nineteen ununited fractures, poor reduction, inadequate mechanical fixation, premature removal of the nail, or a combination of these factors contributed to the non-unions. The remaining nine ununited fractures followed satisfactory reductions and nailings; two of these were in patients with Paget's disease involving the head and neck of the femur.

The number of hips showing aseptic necrosis and degenerative arthritic changes following bony union is shown in Table IV. It is a coincidence that the percentage of aseptic necrosis and arthritic changes in the hip each happens to be 33.6 per cent., as all patients with aseptic necrosis did not have arthritic changes, whereas a few without aseptic necrosis did have arthritic changes. The majority of those with aseptic necrosis had arthritic changes; and, in all probability, all patients with aseptic necrosis will eventually have arthritic changes if weight-bearing is carried out over an appreciable length of time. The incidence of aseptic necrosis seen in patients studied one to two years is essentially the same as that observed in patients studied two years or longer. In the latter group, aseptic necrosis was apparent in the roentgenograms before the elapse of two years. This indicates that aseptic necrosis can be detected within one to two years following the fracture.

The arthritic changes in the head of the femur were classified as 1, 2, 3, and 4, plus. Slight roughening of the head of the femur was rated as 1 plus. A slight depression and irregularity of the head of the femur were classified as 2 plus. Moderate but definite collapse of the head was classified as 3 plus, while marked collapse of the head was classi-

TABLE III

BONY UNION

Age	Union		Non-Union	
	No. of Hips	Per Cent.	No. of Hips	Per Cent.
Under 40	11	100.0	0	0
40–59	31	91.0	3	9.0
60–69	42	78.0	12	22.0
70–79	23	92.0	2	8.0
80 and older	15	88.0	2	12.0
Totals	122	86.5	19	13.5

FIG. 1-A FIG. 1-B

Fig. 1-A: Acute fracture of the neck of the femur in a woman, forty-seven years of age.
Fig. 1-B: Appearance of the bone approximately six months following insertion of the Smith-Petersen nail.

FIG. 1-C

Anteroposterior and lateral roentgenograms, taken nine and a half years following the insertion of the Smith-Petersen nail. The patient has an excellent result, without aseptic necrosis or arthritic changes.

fied as 4 plus. In Table IV, 1 and 2 plus arthritic changes have been listed as moderate; 3 and 4 plus arthritic changes have been listed as severe. Clinically, the patients with moderate arthritic changes have been able to tolerate the pain in the hip for a restricted

Fig. 2-A Fig. 2-B Fig. 2-C Fig. 2-D

Figs. 2-A and 2-B: Lateral and anteroposterior views showing position of the Smith-Petersen nail approximately six months following the fracture of the neck of the femur in a woman, fifty-four years of age.

Fig. 2-C: Appearance of the hip twelve months following insertion of the Smith-Petersen nail.

Fig. 2-D: Appearance of the hip eighteen months following insertion of the Smith-Petersen nail. Arthritic changes are present with beginning collapse of the head.

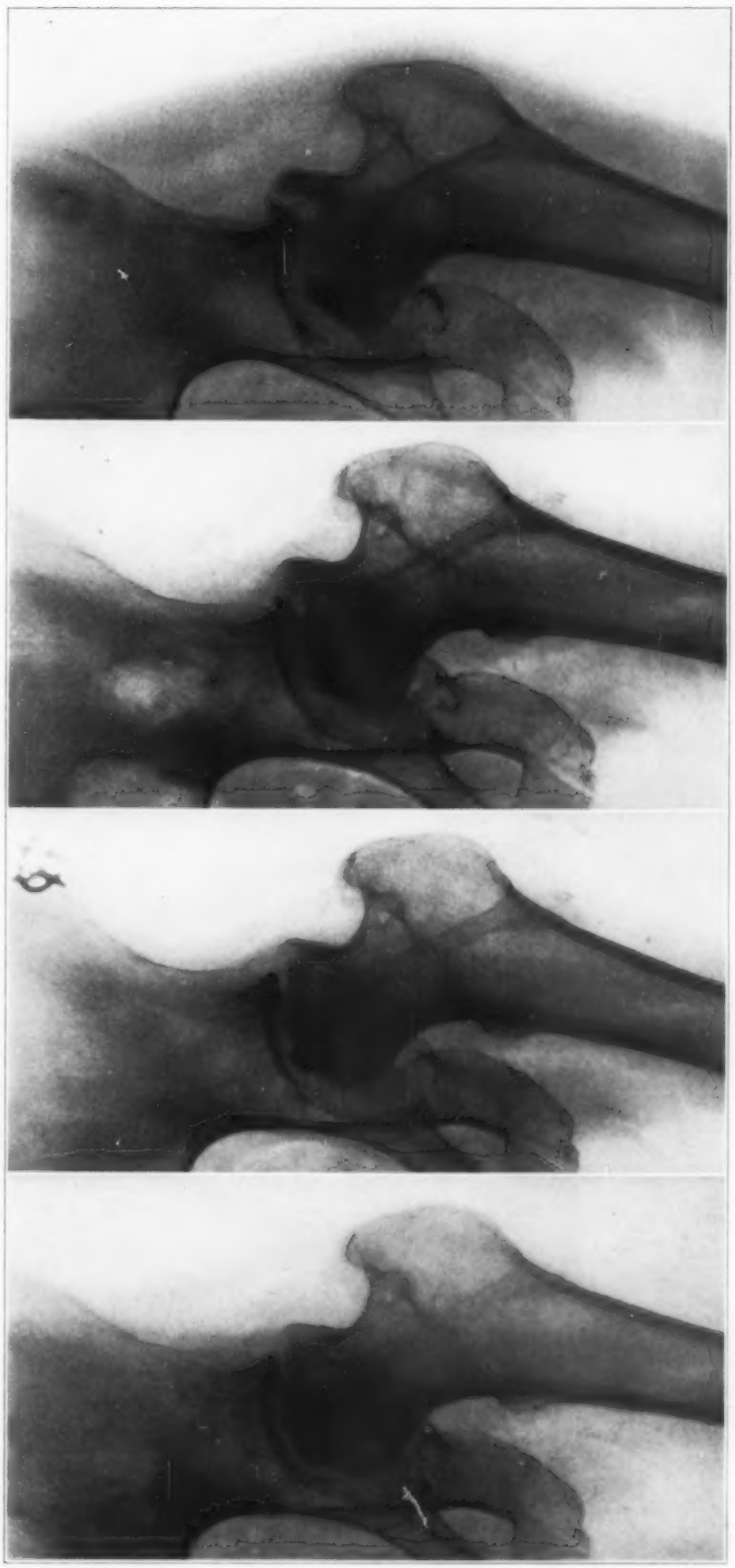

FIG. 2-E FIG. 2-F FIG. 2-G FIG. 2-H

Figs. 2-E, 2-F, 2-G, and 2-H: Appearance of the hip at two years, three years, four years and five years, respectively. These roentgenograms demonstrate a gradually increased collapse of the head, with associated increase in the arthritic changes of the hip joint.

TABLE IV

CHANGES FOLLOWING UNION

Follow-Up Period	No. of Hips	Aseptic Necrosis		Arthritic Changes			
				Moderate		Severe	
		Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
1–2 years	84	28	33.3	13	15.5	6	7.1
Over 2 years	38	13	34.2	10	26.3	12	31.6
Totals	122	41	33.6	23	19.0	18	14.6

TABLE V

END RESULTS

		Per Cent.
Mortality		9.3
Poor End Result		
Non-union	13.5	
Severe arthritic changes	14.6	28.1
Fair End Result		
Moderate arthritic changes		19.0
Good End Result		43.6
Total		100.0

amount of weight-bearing, while those with severe arthritic changes have been definitely handicapped because of pain, especially on weight-bearing.

The final results in this series of patients is shown in Table V. According to these findings, the average fracture of the neck of the femur has the following prognosis: mortality, 9.3 per cent.; poor end result, 28.1 per cent., including ununited fractures, and severe arthritic changes; a fair end result, 19 per cent.; and a good end result, 43.6 per cent.

The data presented in this paper indicate that the total number of patients with severe arthritic changes in the hip will exceed the number with non-union. In the future, the problem of rehabilitation and treatment of the patients with severe degenerative arthritic changes may become more important than the treatment of those with ununited fractures.

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NOTE: Discussion of this paper and of the other papers of this Symposium will appear in the April issue of *The Journal*.

Editor

THE PATHOLOGY OF UNUNITED FRACTURES OF THE NECK OF THE FEMUR * †

BY MARY S. SHERMAN, M.D., AND D. B. PHEMISTER, M.D., CHICAGO, ILLINOIS

From the Department of Surgery, University of Chicago

That the incidence of non-union is relatively high in intracapsular fractures of the neck of the femur has long been known. More recently, considerable work has been done towards explaining the reasons for this and its prevention. It is now generally agreed that the principal factors which determine whether union or non-union will occur are the extent of original injury, the presence or absence of impaction, the accuracy of reduction and fixation, the survival or death of the femoral head, and the fact that no peripheral callus is laid down because of the absence of a cambium layer on the femoral neck. Although it is well known that union can occur in the presence of aseptic necrosis of the head, still non-union is much more frequent when the head dies than when it survives[11]. It has also become clear that whether or not union occurs, pathological changes in the hip joint are rare if the head is alive, but are constant and may become severe if the femoral head is dead[14]. This paper will attempt a review of the pathological changes seen, following ununited fractures of the femoral neck.

Whether the femoral head survives the fracture depends, of course, on how much the blood supply was damaged; there is no evidence that death occurs long after the fracture when union has failed. There have been several excellent papers which have described in detail the circulation of the femoral neck and head[4,7,9,17]. It is known that the chief supply to the head is through branches of the anterior and posterior circumflex vessels which lie in that portion of the capsule which is reflected up on the neck, and more especially in the posterior portion of the capsule. If this portion remains intact, as it not infrequently does, there may be sufficient circulation to preserve the head. There is also a variable amount of the head which is supplied through the ligamentum teres[4,9,16]. The distribution of these two sets of vessels and the amount of collateral circulation between them vary greatly, but the extent of bone supplied by end arteries may be great. If the neck and capsule are completely severed and the round ligament carries no blood supply, the entire proximal fragment dies.

If the head has remained viable, but non-union nevertheless supervenes, the principal additional change is disuse atrophy which is approximately equal in both fragments. The head never collapses, and it is especially noteworthy that the articular cartilage is well preserved. Operative procedures designed to produce union are usually successful, and degenerative arthritis does not follow. This is illustrated by the following three cases.

CASE 1. M. D., a woman, forty-five years old, whose fracture, treated by skeletal traction and an abduction cast, showed no evidence of union four months later (Fig. 1-A). The roentgenograms revealed the uniform atrophy of both proximal and distal fragments, which is indicative of survival of the head. At operation the entire ununited area was fixed with a tibial peg. A biopsy specimen removed from the head showed living bone. Five months after the operation, healing was well established, and the patient was ambulatory. Two years later, the patient was still asymptomatic, and the roentgenogram showed a cartilage space of normal width and no evidence of degenerative changes in the joint (Fig. 1-B).

CASE 2. F. G., male, aged sixty, was admitted to the Hospital on February 18, 1922, with an ununited intracapsular fracture of the neck of the femur of fifteen months' duration. A roentgenogram (Fig. 2-A) revealed extensive loss of the neck fragments and regional reduction in bone density which was equally marked in the acetabulum, the head, and the distal fragment of the femur. The shadow of the cartilage

* Presented at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 29, 1946.

† This work was aided by a grant from the Douglas Smith Foundation.

FIG. 1-A

FIG. 1-B

Fig. 1-A: Case 1. Roentgenogram four months after fracture of left femoral neck shows no evidence of healing. Note uniform atrophy of both fragments, indicating a viable head.

Fig. 1-B: Roentgenogram two years after insertion of tibial peg shows union. The head and the rest of the femur are still of the same density, and there is no evidence of degenerative changes in the joint.

space of the joint was of normal width. The uniform reduction of density is indicative of a living atrophic head. A Whitman reconstruction operation resulted in good function (Fig. 2-B).

There was a pseudarthrosis between the two shortened fragments of the femoral neck, which were connected in places by fibrous adhesions. The articular cartilage of the excised head was smooth and normal in external appearance. Only a small part of the proximal fragment of the neck remained, and its eroded fracture surface was covered by fibrous callus bordering on the false joint space. Section of the head showed the articular cartilage to be of normal thickness. There was marked osteoporosis of the bone, which was rich in fatty marrow.

A microscopic section was made of the lower half of the head, extending from the fovea to the fracture line (Fig. 2-C), which disclosed interesting changes: The trabeculae were greatly reduced in size and number, but their cells were alive. The bony cortex, which was covered by an almost normal-appearing articular cartilage, was greatly thinned (Fig. 2-D). The surfaces of the trabeculae were in many places

FIG. 2-A

FIG. 2-B

Fig. 2-A: Case 2. Fifteen-month-old ununited fracture of neck. Equal degree of atrophy in both fragments is indicative of a living head.

Fig. 2-B: Three years after Whitman reconstruction operation.

covered by mononuclear cells, some of which were still actively engaged in the process of smooth absorption of bone. The marrow consisted principally of fat, and there was only a small amount of hemopoietic tissue. Near the base of the head there was an island, about one-half centimeter in diameter, of fibrous marrow. New bone had been laid down between the trabeculae along the fracture line to form a cortex which was covered by fibrous tissue with an irregular synovial lining along the surfaces of the false joint. There was no callus along the external surface of the cortex of the neck.

The diagnosis was ununited fracture with pseudarthrosis of the neck of the femur and atrophy of disuse of the viable head.

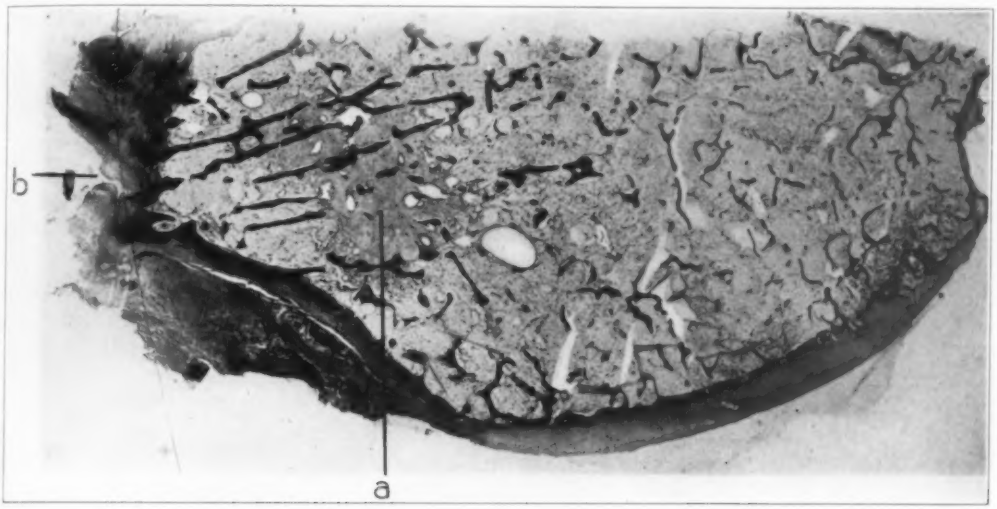

FIG. 2-C

Section through the head from fracture line to fovea, showing atrophic bone with fatty marrow except for fibrous area (*a*) and lining of pseudarthrosis (*b*).

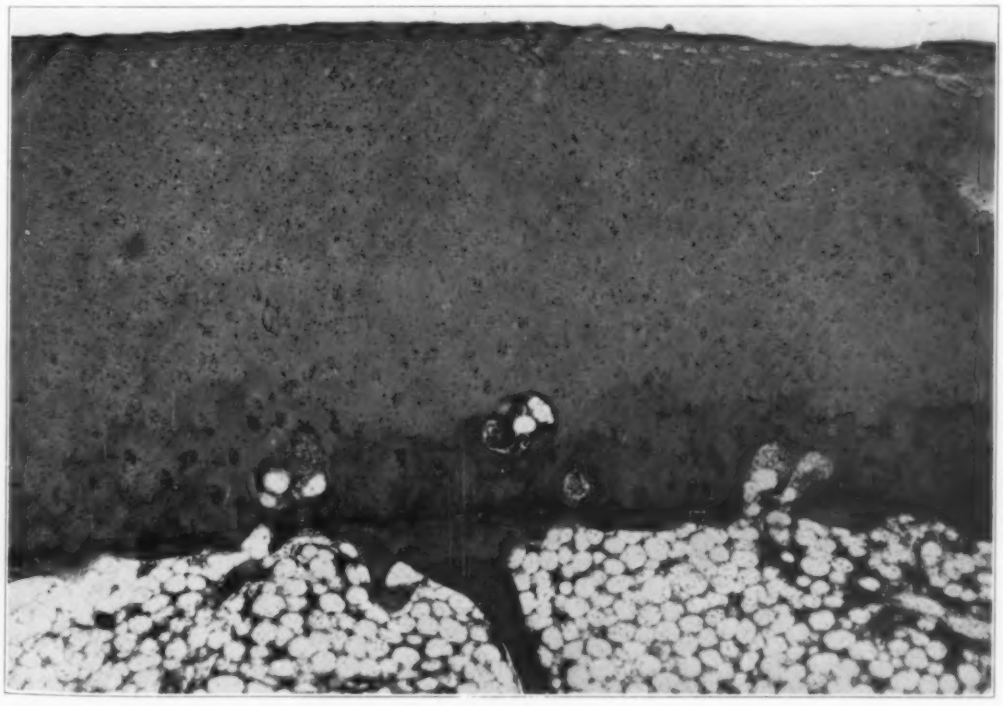

FIG. 2-D

Articular cartilage is changed very little. Bony cortex and trabeculae are greatly reduced in size, but cells are alive. Living fatty and hemopoietic marrow is evident.

CASE 3. A. P., a woman, aged sixty-three years, entered the Hospital with an ununited intracapsular fracture of the neck of the right femur of three and one-half months' standing, which had resulted from slight trauma seven months after x-ray treatments for carcinoma of the cervix uteri. Two roentgenographic examinations during this period showed the hips to be normal.

A roentgenogram at the time of admission (Fig. 3-A) revealed osteoporosis of the bones of the right

FIG. 3-A

FIG. 3-B

Fig. 3-A: Case 3. An ununited fracture of the neck of the femur, of three and one-half months' duration, which occurred seven months after roentgenotherapy for carcinoma of the uterus. The head is alive.

Fig. 3-B: Two years after operation.

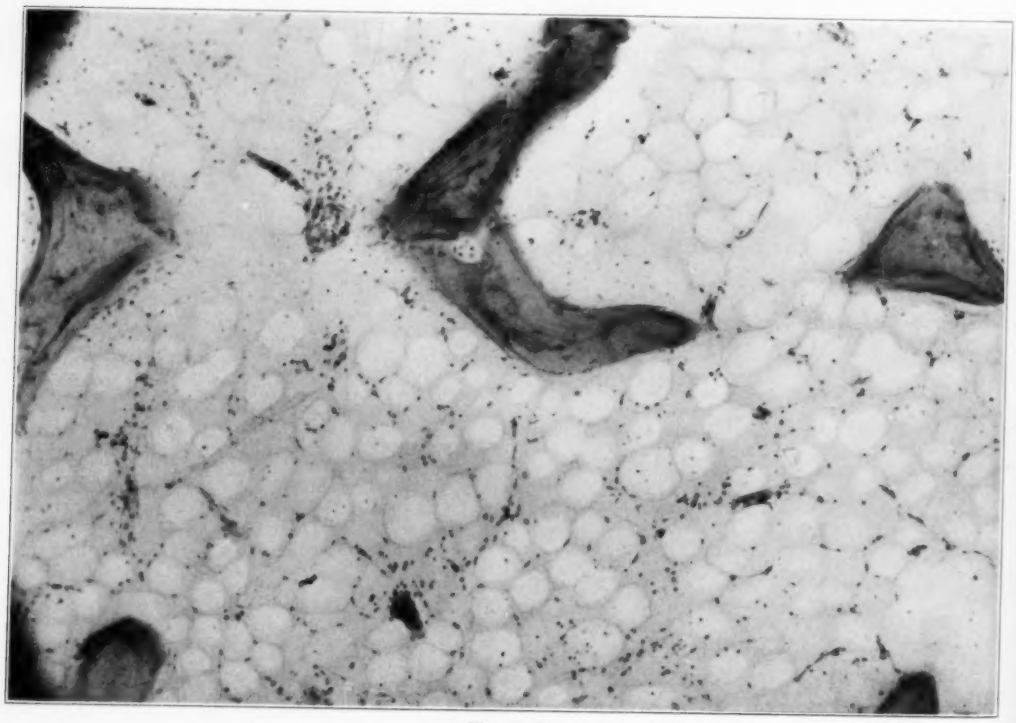

FIG. 3-C

Atrophic living bone of head.

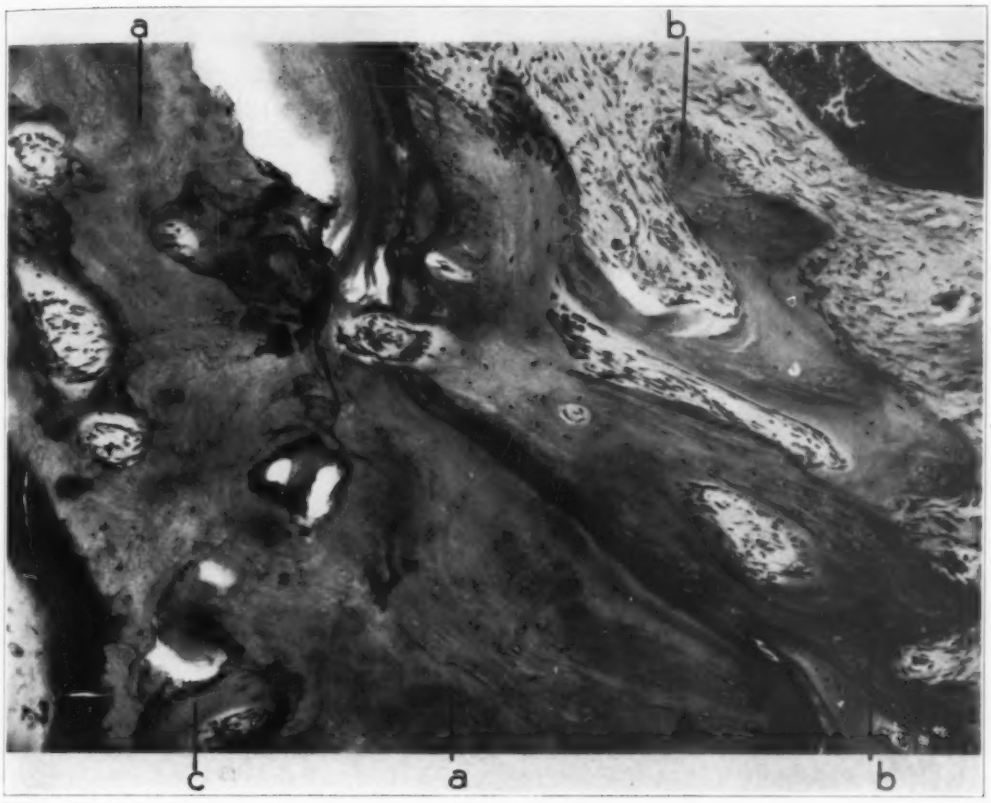

FIG. 3-D

Specimen from edge of neck fragment at fracture margin, showing necrotic trabeculae (*a*), fibrous and bony callus (*b*) of the distal fragment, and trabeculae undergoing creeping replacement by new bone (*c*).

hip region, a subcapital fracture of the neck with upward displacement of the lower fragment, and equality of density of the bone of the two fragments. The opposite hip, which had also been irradiated, showed generalized osteoporosis.

At operation the fracture was reduced and held in position by three heavy threaded wires, and four small grafts of non-irradiated bone were onlaid across the fracture line. Biopsy specimens were taken from the head and from the ends of the neck fragments.

Prompt union followed, and the function of the hip was restored to almost normal. A roentgenogram made two years after operation (Fig. 3-B) showed the density of the pelvis, head, and distal fragments to be about equal and even throughout; the contour of the head and the outlines of the joint space had been preserved, all of which indicates that massive necrosis of the head was not produced either by the irradiation or by the fracture of the neck.

The bone from the head was spongy, and free bleeding occurred during its removal. Microscopically the trabeculae were greatly reduced in size, but the lacunae contained cells, the great majority of which stained normally (Fig. 3-C). The cancellous spaces were filled with fat, throughout which were scattered small islands of hemopoietic cells. The overlying articular cartilage was alive, but showed some fibrocartilage proliferation along the surface at the margin of the head.

Microscopic sections of the specimens removed from the end of the neck fragments revealed a mixture of dead and living trabeculae with fibrous and bony callus in the cancellous spaces and along the fracture surfaces (Fig. 3-D). It is impossible to state how much of the necrosis was due to the radiation and how much to the fracture.

The diagnosis was post-irradiation osteoporosis of the bones of the hip, and ununited fracture of the neck of the femur with survival of the head.

Badgley and Bowman have studied twenty-eight fractures of the neck of the femur, occurring after irradiation for malignancy located in the female genitalia. Of these fractures, eighteen were not treated, seven of which failed to unite. The authors cite the report

of six cases, including three of their own, in which microscopic studies were made of the bone of the femoral neck. There was marked osteoporosis, increase in volume of fatty marrow, and sclerosis of the nutrient vessels. The fibrous callus was avascular, and there was a lack of osteogenesis throughout the fracture area. Minute fragments of dead bone were found in the fibrous tissue. From roentgenographic appearances, it was believed that the head always remained alive[1].

When the fracture disrupts the blood supply to such an extent that the head dies, atrophy of disuse cannot occur. Therefore, as atrophy of the still-living surrounding bone proceeds and the head remains unaltered, it stands out as relatively more dense. Gradually there is invasion of the necrotic portion by blood vessels and connective tissue, which grow in from the edges of the capsule and from the ligamentum teres. This tissue slowly absorbs the old dead trabeculae and at the same time lays down cancellous new bone on other surfaces and in between the trabeculae. This new bone is less dense than the old bone, and thus the roentgenogram of a partially transformed head shows variable density. The necrotic articular cartilage does not show much change until a blood supply reaches it, either from the revascularized underlying bone or from areas such as the fovea. Then it is gradually replaced, either by an imperfect fibrocartilage surface or even in spots by bone. The degenerative changes which follow may be few, if replacement has occurred rapidly and there has been relatively little breakdown. If the reparative process is slow, the articular cartilage wears away, and pieces may break off to form loose bodies. The joint surfaces are badly damaged, and the subsequent osteo-arthritis may be severe.

Case 4. W. H., a farmer, fifty-seven years old, had been an invalid for three years and bedridden for six months because of amyotrophic lateral sclerosis. A fall resulted in a fracture of the neck of the left femur which, to facilitate nursing care, was fixed with three threaded steel wires. Ten days later, when the fracture was twenty-two days old, the patient expired of pneumonia, and the upper end of the femur was obtained at postmortem examination.

The fracture through the medial portion of the neck was complete, and the two fragments were connected only by the anterior inferior capsule, which remained intact. On section, the bone of the head

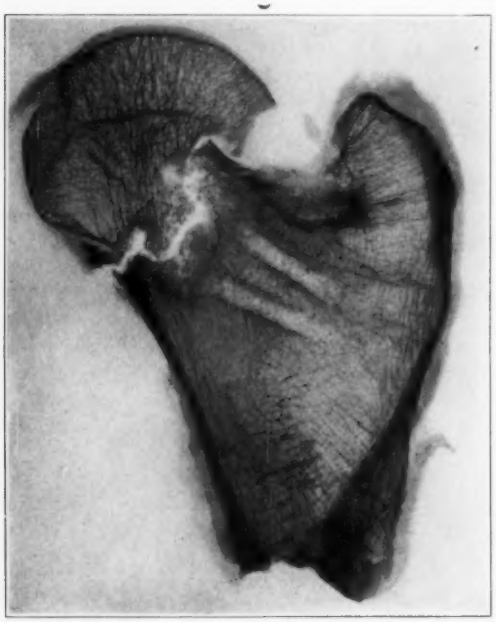

Fig. 4-A

Case 4. Roentgenogram of slice of specimen shows greater atrophy of the distal fragment than of the femoral head, which is necrotic.

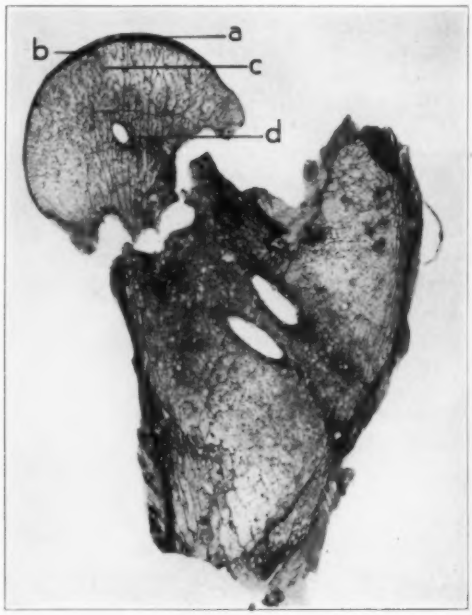

Fig. 4-B

Microscopic section of specimen shows greater atrophy of the distal fragment. The letters mark the areas from which the photomicrographs were taken.

appeared whitish-yellow, except for a small area around the fovea which was pink. The roentgenogram of the slice cut from both fragments (Fig. 4-A) showed so much old atrophy of disuse throughout that it was difficult to be sure of the nutritional state of the head, but its trabeculae appeared perhaps a little denser than those of the neck and trochanteric regions of the distal fragment. In the large microscopic section, this greater amount of atrophy in the distal fragment was more obvious (Fig. 4-B).

On microscopic examination, the articular cartilage was found to be intact, except along the joint surface where it showed degenerative changes. The lacunae were not seen, the nuclei were shrunken and pyknotic, and the staining was blotchy (Fig. 4-C). The trabeculae of the head were entirely necrotic, except for a small zone around the fovea. In this area the osteocytes were clearly seen, and the normal marrow elements were preserved. In the necrotic portion immediately adjacent to the fovea (Fig. 4-D), creeping replacement of dead marrow and trabeculae had already begun, and new bone was being laid down. Elsewhere there remained no normal marrow, and many of the spaces were filled with amorphous necrotic debris. In places where the vessels remained intact, there was a different picture. The vessels themselves were markedly engorged. The fat cells had survived, and occupying the spaces between them and the swollen vessels there were great numbers of huge pale macrophages, many of which appeared vacuolated or foamy (Fig. 4-E).

The tract of one wire, which was included in the section, was lined with a very vascular fibrous tissue. There was active membranous ossification in this fibrous tissue and rapidly advancing replacement of the dead trabeculae in the walls (Fig. 4-F). The same osteogenic activity was seen in the lower portion of the head, next to the fracture line. The blood supply must have been preserved by, or re-established from, the untorn capsule of this region.

Case 5. R. S., male, aged fifty-seven years, entered the Hospital two days after injury, with an impacted subcapital fracture of the right femoral neck (Fig. 5-A). The limb was immobilized in a plaster spica for two months. A roentgenogram at the end of that time showed healing of the fracture with no apparent density differences between head and distal fragment. The patient walked on crutches until six and one-half months after injury, when a roentgenogram (Fig. 5-B) revealed unaltered density of the head and blotchy shadows of reduced density, proximal to the fracture line in the neck, suggestive of death of the proximal fragment with creeping invasion and replacement by less dense new bone. There was relatively little pain or restriction of motion in the hip. Thereafter, the patient walked with a cane, and one year following injury had only a small amount of weakness and discomfort in the hip. A roentgenogram at that time showed changes similar to those seen in Figure 5-B, and the patient resumed work as a painter. Seventeen months after injury he returned, stating that the hip had recently become more painful.

A roentgenogram (Fig. 5-C) showed a broad zone of blotchy density across the subcapital portion of the neck. There was a slight depression of the cortex of the top of the head where it receives the weight from the acetabulum. The small undepressed area of the head lateral to the superior acetabular margin was irregularly reduced in density, suggesting replacement by new bone. This slight depression of the unaltered weight-bearing portion was indicative of beginning collapse of the untransformed portion of a necrotic head.

The hip steadily grew worse and a roentgenogram nineteen months after injury (Fig. 5-D) showed an irregular fracture line along the base of the head with definite downward slipping of the head and increased density in the neck distal to the new fracture line.

A diagnosis was made of a pathological fracture at the junction of the dead and the transformed living portions of the head and neck.

At operation the head and medial portion of the neck in the region of the old healed fracture were resected, and tibial-bone transplantation for fusion of the hip was carried out.

In both roentgenogram and microscopic section of a mid-sagittal slice of the specimen (Fig. 5-E), the line of the recent pathological fracture was clearly seen. The trabeculae of the head fragment were even in density and appeared unaltered. Microscopically, the structures proximal to the new fracture line consisted of old dead bone and articular cartilage, except below the fovea where both articular cartilage and cortex had been invaded by blood vessels from the round ligament and replaced by cancellous new bone. Distal to the new fracture line, the structure consisted almost entirely of living bone, which had repaired the original fracture that interrupted the blood supply of the proximal fragment and had grown into and replaced the old dead bone, except for portions of the heavier trabeculae that were still in the process of being replaced.

The new fracture line passed through the zone of invasion and replacement of the dead bone by living bone, as shown in the photomicrograph made through its lower one-third (Fig. 5-F). The uninvaded bone of the head (*a*) consisted of trabeculae that were unaltered, except that their cells were gone, and of dead marrow with the outline of the fat cells preserved. The tissue along the invading front (*b*) consisted of connective tissue and blood vessels which had replaced the dead marrow, but had produced little absorption of the dead trabeculae. In the deeper layer, the trabeculae showed evidence of absorption, and in some places, of replacement by spongy new bone. It was in this weakened zone that the

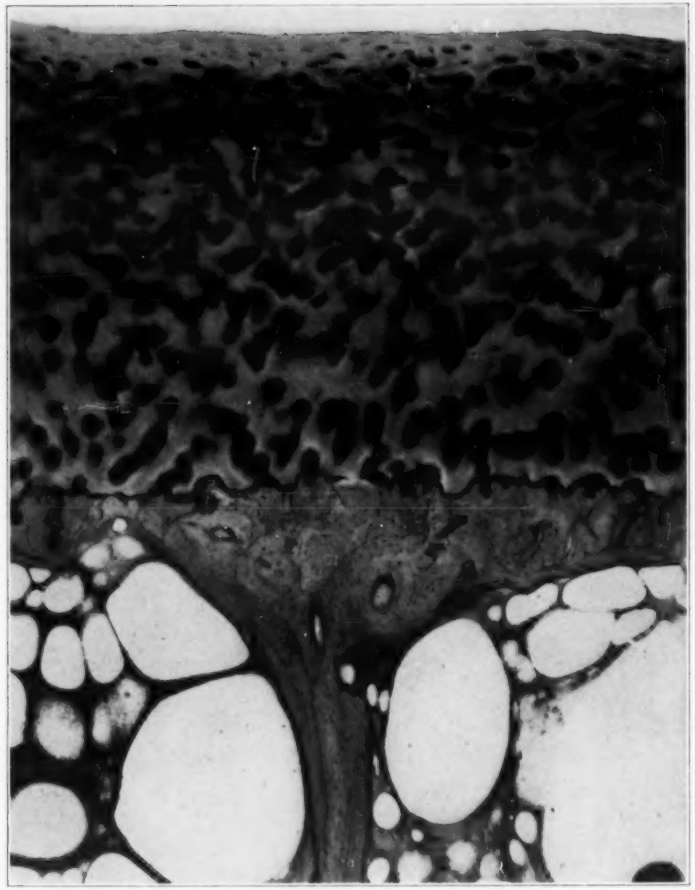

FIG. 4-C

Fig. 4-C: Specimen showing necrotic bone, marrow, and articular cartilage. There has not yet been time for much change to take place in the surface.

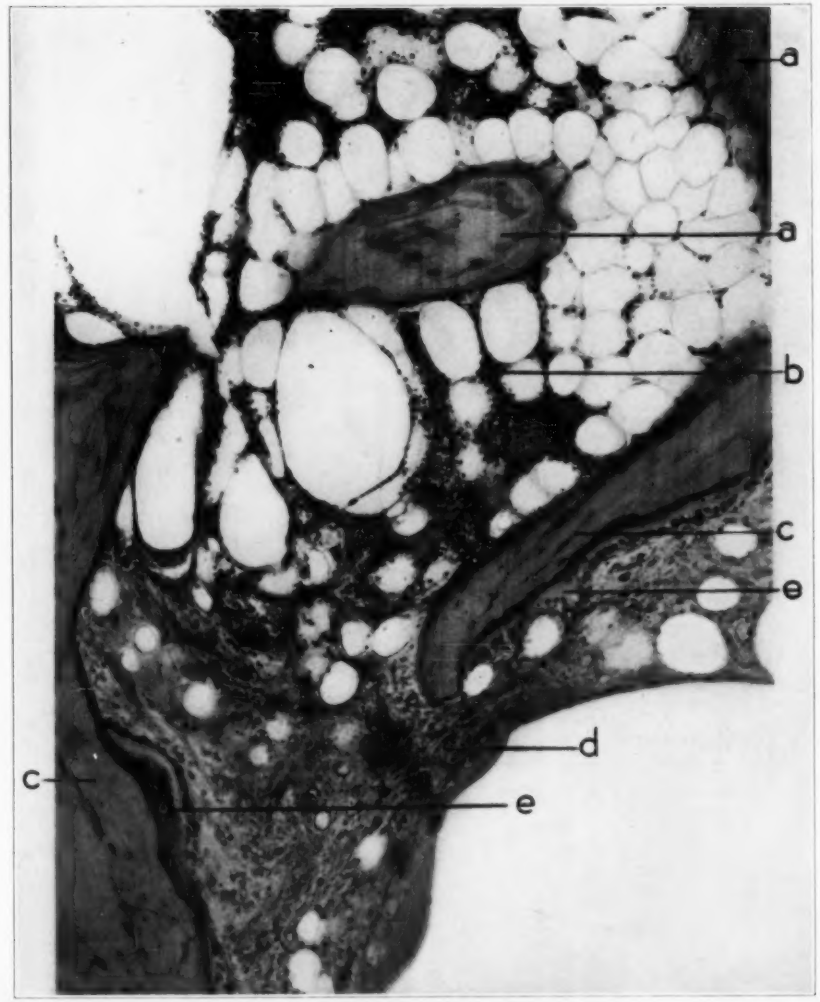

FIG. 4-D

Fig. 4-D: Specimen from area of fovea at boundary of living bone and marrow (*a*, *b*) and necrotic bone (*c*), which is already being replaced by new living bone (*e*). Necrotic marrow is being replaced by vascular fibrous tissue (*d*).

in the surface.

Fig. 4-D: Specimen from area of fovea at boundary of living bone and marrow (*a*, *b*) and necrotic bone (*c*), which is already being replaced by new living bone (*e*). Necrotic marrow is being replaced by vascular fibrous tissue (*d*).

FIG. 4-D

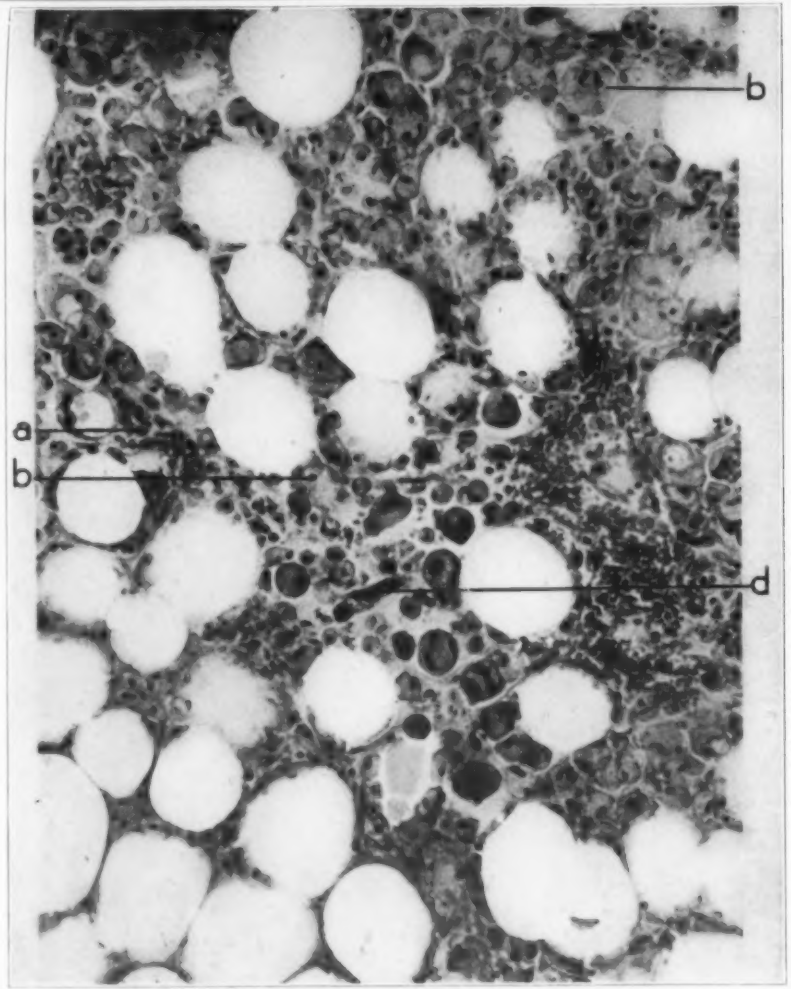

FIG. 4-E

Scattered among the fat cells are engorged blood vessels (*a*) and many swollen, fat-filled macrophages (*b*).

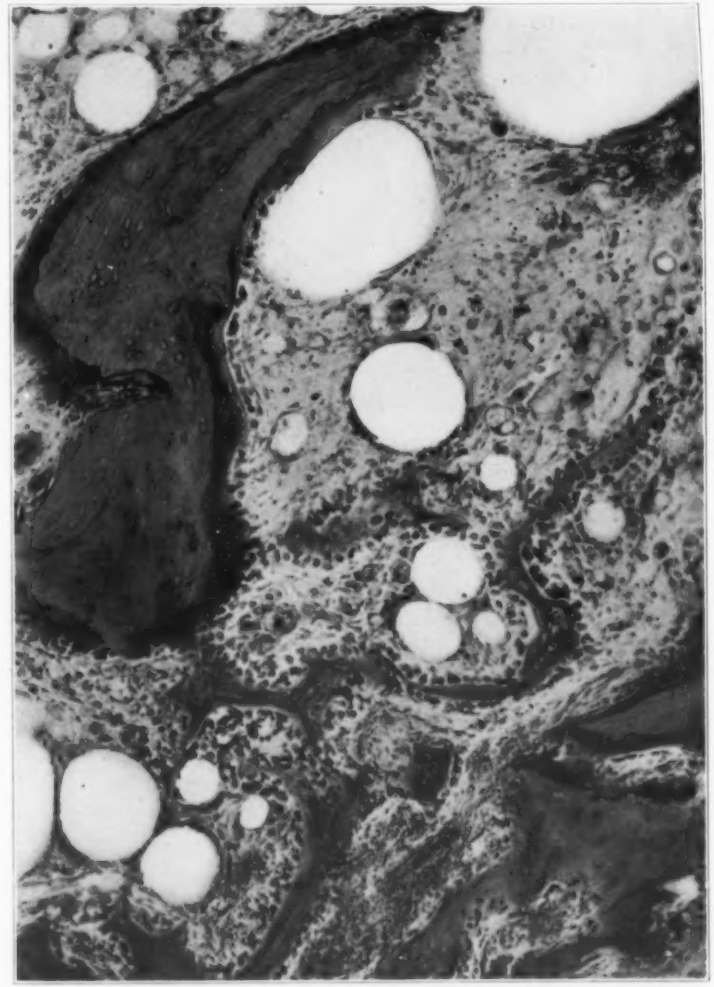

FIG. 4-F

Necrotic trabeculae are covered with a layer of new living bone. In the vascular fibrous marrow there is active membranous ossification.

fracture had occurred. Distal to the fracture, the trabeculae had been largely replaced by living bone, and the marrow consisted of fat, fibrous tissue, and hemopoietic elements. The fracture line was filled with fibrous callus and numerous small fragments of broken trabeculae. There was newly formed bone in some of the callus, and lacunar absorption of the fragments of trabeculae, as seen in Figure 5-G.

Pathological fracture through the junction of the dead and living bone from prolonged weight-bearing is common in aseptic necrosis of the head from any cause[2]. However, it is rare to see almost the entire head sheared off as is the case here. This is because the upper portion is the last to be replaced; and, by the time it has been weakened by invasion, the

FIG. 5-A

FIG. 5-B

Fig. 5-A: Case 5. Recent impacted subcapital fracture of the neck of the femur.
Fig. 5-B: Six and one-half months after injury. Fracture united. The unchanged density of the head and the blotchy reduction in density of the neck proximal to the fracture site suggest death of the proximal fragment and beginning creeping replacement of dead bone by new bone.

FIG. 5-C

FIG. 5-D

Fig. 5-C: Seventeen months after fracture. Beginning depression of the weight-bearing portion of the head.
Fig. 5-D: Nineteen months after original injury. Recent pathological fracture with downward displacement of the remaining dead portion of the head, produced by weight-bearing on the weak zone of invasion and replacement.

lower portion has already become strong enough to withstand weight-bearing. Therefore, usually only the upper part of the head breaks off and collapses. However, delayed union or non-union between the dead and living portions is the rule[6,11] and in time the collapsed dead bone of the top of the head may even be absorbed and replaced by fibrous tissue instead of bone.

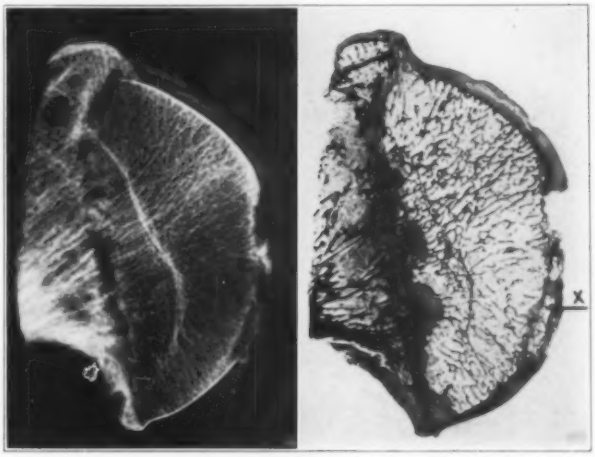

FIG. 5-E

Roentgenogram (left) and stained section (right) of slice of the excised head and part of the neck, showing recent pathological fracture between distal transformed and proximal necrotic portions.

CASE 6. W. G., a man of fifty-six, had injured the left hip while bowling two years before his admission. He had enough pain to keep him in bed for ten weeks, at which time a roentgenogram showed a fracture of the left femoral neck. The fracture was reduced and fixed with three Steinmann pins. These were removed seven weeks later, and the patient even returned to work, although he had constant pain and moved with difficulty. A roentgenogram made two and one-half years after injury showed non-union of the fracture (Fig. 6-A). The femoral head appeared denser than the surrounding bone and there were very few areas of decreased density, which indicates little replacement. The necrotic femoral head and two well-developed loose bodies were removed and a hip fusion was performed with a tibial graft. The fusion was clinically solid nine months

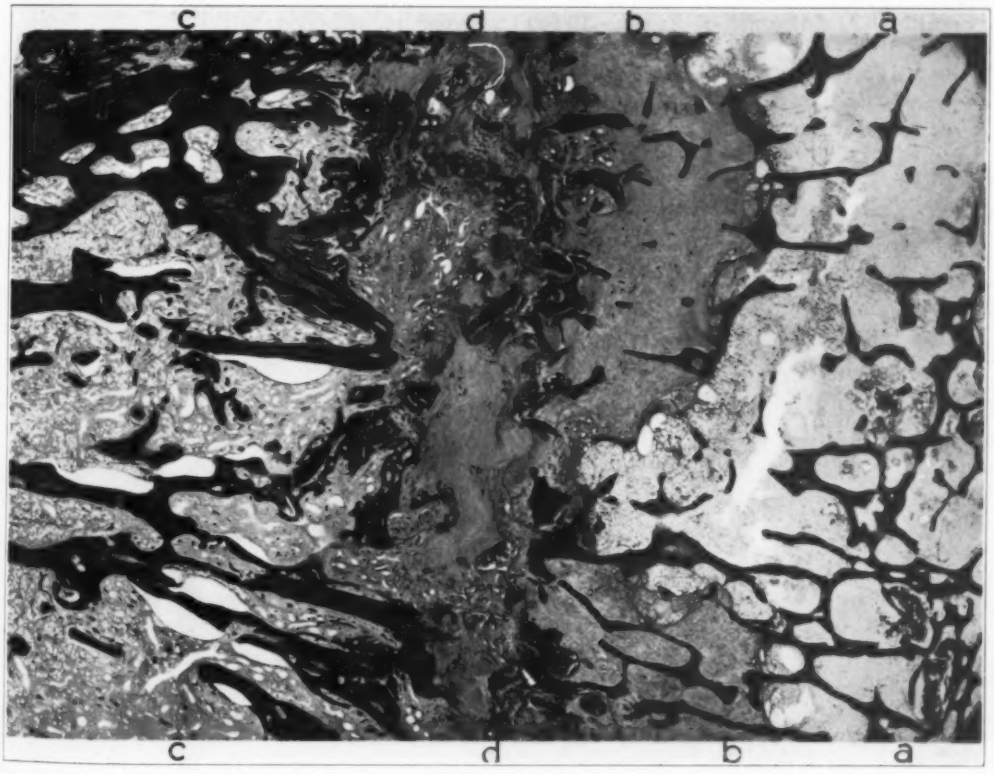

FIG. 5-F

Zone of invasion and replacement of dead bone by living bone, and fracture line through its weakened portion. *a–a* is the uninvaded dead head; *b–b*, the front of the invading tissue; *c–c*, the replaced living bone; and *d–d*, the new fracture line.

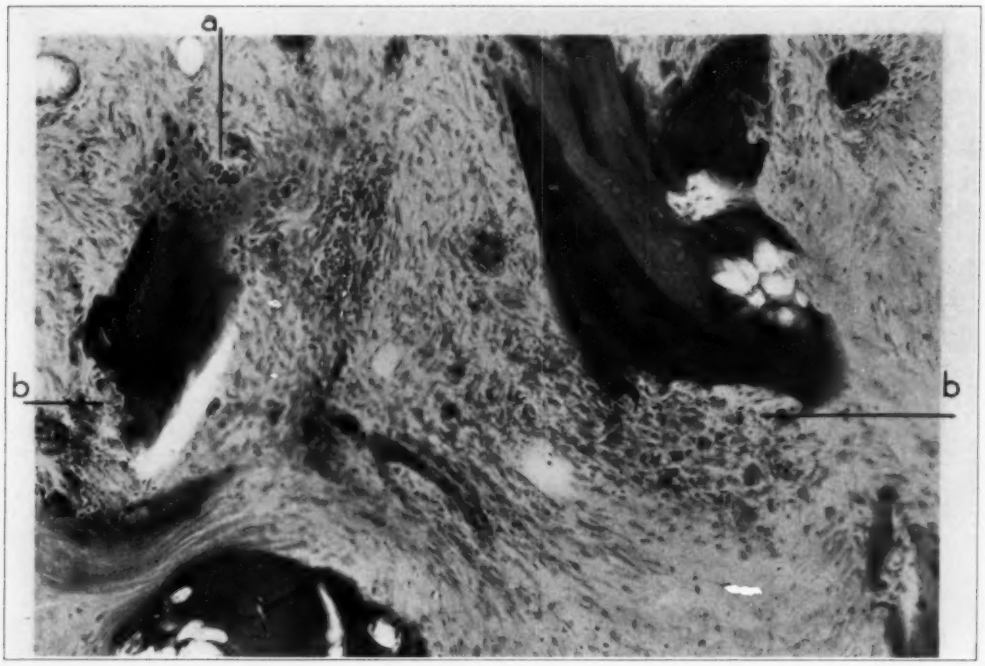

FIG. 5-G

The fracture line (× 150), showing fibrous callus with newly formed bone (*a*) and lacunar absorption of fractured necrotic trabeculae (*b*).

after operation. A photograph of the excised head shows the marked degeneration of the articular surfaces (Fig. 6-B).

A roentgenogram of a sagittal slice of the head demonstrates the great difference in density in different areas (Fig. 6-C), and the reason for this is seen clearly in the section (Fig. 6-D). The upper weight-bearing portion of dead bone shows no tendency towards revascularization. The articular cartilage and all the trabeculae are necrotic, and the marrow spaces are filled with an amorphous, pink-staining debris (Fig. 6-E). At the upper edge, this debris, in which appear nodules of living fibrocartilage, several of which surround bits of dead bone, has separated the articular cartilage from the bone below. The central portion of the head, which corresponds to the "cystlike" areas seen on the roentgenogram of the slice, exhibits, in addition to necrotic cartilage and trabeculae, marrow spaces which are entirely filled by a dense mature fibrous tissue. This tissue is quite inactive and in most regions there is an absolute lack of osteoblasts and osteoclasts, showing that repair has come to a standstill (Fig. 6-F). However, in the region of the fovea and also at the fracture edges, where weight-bearing is not a discouraging factor and where blood vessels are abundant, there is evident osteogenic activity and creeping substitution has begun (Fig. 6-G). Figure 6-H shows a section of one of the osteocartilaginous loose bodies.

CASE 7. J. G., a seventy-year-old man, had been an inmate of the county poor farm for two years before he died of bronchopneumonia. There was no record as to the clinical condition of the right hip. At postmortem examination an ununited fracture of the right femoral neck was found, and the entire hip joint was removed.

Although there was no evidence of union of the fracture fragments, the head was firmly bound both to the remnants of the neck and to the acetabulum by dense fibrous adhesions. On the cut surface of the sliced specimen there was noted a large area of bone in the mesial portion of the femoral head bordering on the superior articular cortex. This bone was white, and its trabeculae were much heavier than those of the surrounding portions, from which it was separated by a narrow strip of pearly-gray fibrous tissue. In the porous bone immediately surrounding this area, there was a brownish zone of marked vascularity. A roentgenogram of the slice showed complete absorption of the neck with marked sclerosis of the surfaces bordering on the ununited fracture line (Fig. 7-A). There was severe degeneration of the joint surfaces, and the roentgenographic changes are indicative of the partial replacement of a necrotic head. Substitution had proceeded up from the fracture line and inferiorly along the capsular attachments, so that a portion of dense bone had been surrounded by bone of reduced density and was clearly outlined. A separate little island of replaced bone could be seen in the region of the old fovea. The same areas can be identified grossly on the large microscopic section (Fig. 7-B).

Microscopically these areas were composed of trabeculae which had been almost completely replaced by spongy living bone, except for a few persisting central cores of necrotic bone. Normal fatty marrow was present. Within the dense portion both bone and marrow were completely necrotic, and there was no evidence of replacement (Fig. 7-C). The boundary between living and dead bone was clearly marked by a narrow zone where dense collagenous fibrous tissue filled the marrow spaces, but where there was absolutely no osteogenic activity. In many places, where advancement of the process had apparently come to a halt, there was calcification in the fibrous tissue. Immediately beneath the fibrous tissue was vascular living marrow at the edge of the advancing replacement zone (Fig. 7-D).

The articular cartilage overlying the dead central portion of the head was necrotic, except where a thin layer of fibrocartilage had grown over the surface. Over the foveal area of living bone the cartilage, though imperfect, had been revitalized. From the edges of this island, blood vessels had extended into the adjacent dead tissue and along them calcification and even ossification within the necrotic articular cartilage were taking place (Fig. 7-E). On both sides of the dead portion of the head, where replacement had proceeded right up to the cortex, the articular cartilage appeared to be living (Fig. 7-F).

On the acetabular side of the joint the changes were also spectacular. There was nowhere any normal articular surface. At both edges there was very imperfect fibrocartilage formed over bony spurs which grew over the old surface. Remnants of the original articular cartilage could be seen four or five millimeters back in the bone. There was no trace of the original bony articular cortex. Such marked changes in the articular surface of the acetabulum were commonly seen in old cases of ununited fractures of the femoral neck with death of the head[12,13].

FIG. 6-A

Case 6. Two and one-half years after fracture of the left femoral neck. The head is denser than the rest of the femur. The small area of decreased density at the lower edge indicates replacement.

It is noteworthy that degenerative arthritis is regularly present in long-standing cases of necrosis of the head with non-union,

FIG. 6-B

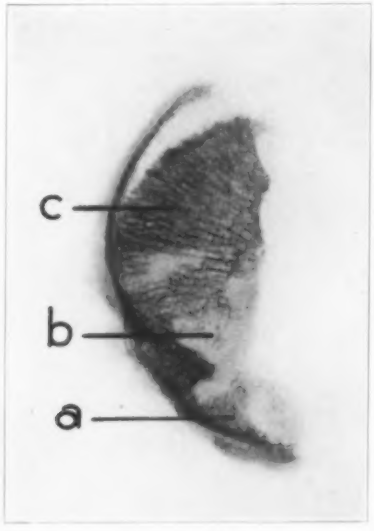

FIG. 6-C

Fig. 6-B: Photograph of the excised femoral head shows marked degeneration of the articular cartilage, which in places has been worn completely away.

Fig. 6-C: Roentgenogram of a slice of the head shows replacing new spongy bone (*a*), dead bone invaded by fibrous tissue (*b*), and uninvaded dead bone and marrow covered by necrotic articular cartilage (*c*). The separation of the cartilage at the top is an artefact.

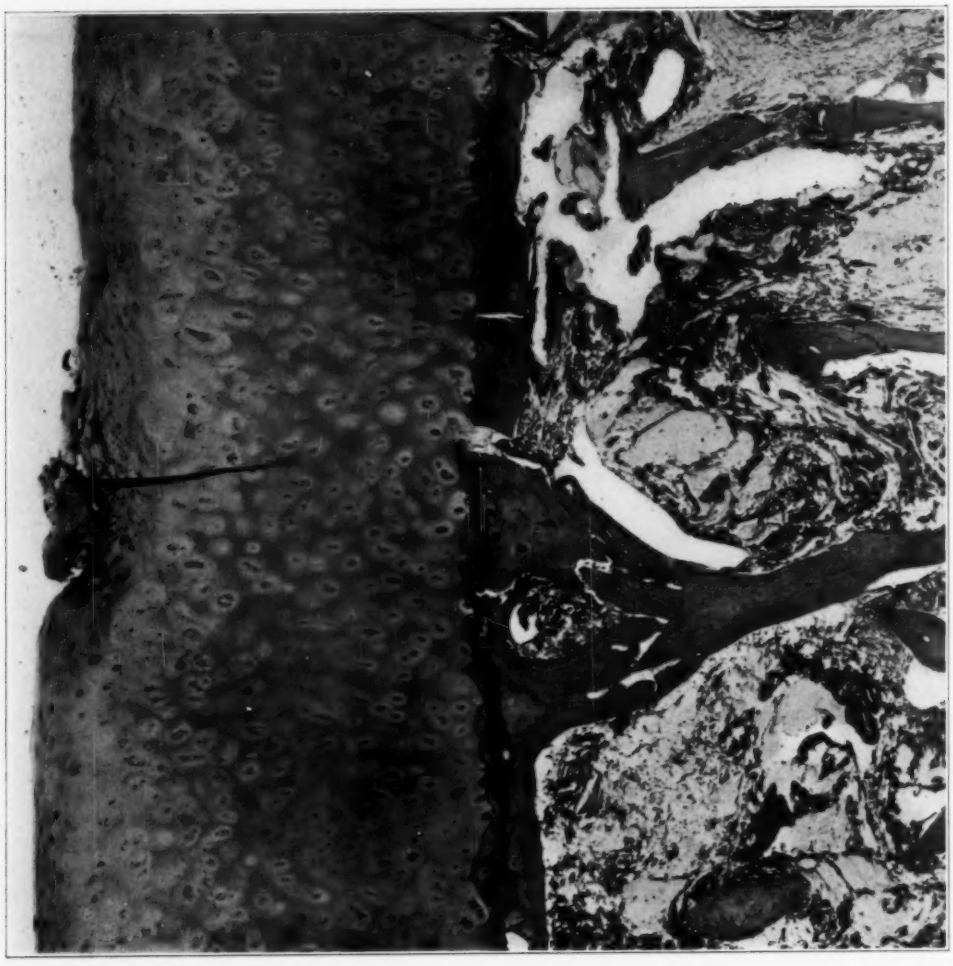

FIG. 6-E

Necrotic bone from upper part of head (*a* in Fig. 6-D) with marrow spaces filled by amorphous debris. Beginning degeneration of dead articular cartilage is evident.

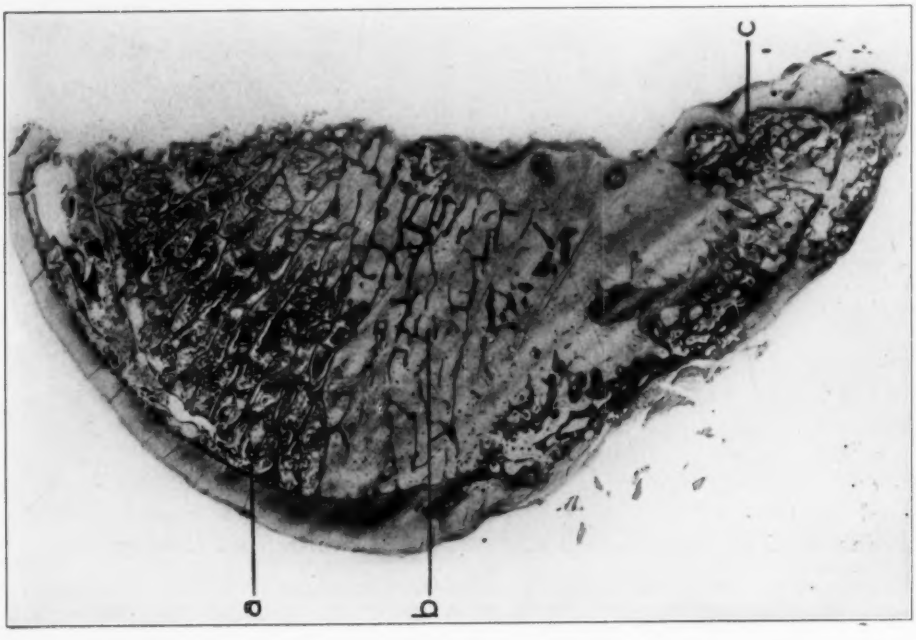

FIG. 6-D

Microscopic section of a sagittal slice of the head. The entire head is necrotic, except for a small area at the bottom which has been replaced by new bone. In the center there is fibrous tissue which is absorbing, but not regenerating, bone. The upper weight-bearing portion has not even been invaded by fibrous tissue. Letters refer to photomicrographs (Figs. 6-E, 6-F, 6-G, and 6-H).

but it is rarely as severe as in old cases of necrosis of the head with bony union. When there is non-union of the fracture, there is usually no collapse of a necrotic head; but, if the fracture unites in the presence of a dead head and weight is borne, collapse of the partially replaced head is to be expected.

The treatment of ununited fracture of the neck with death of the head by open reduction and fixation of the fragments may be followed by bony union. Under favorable circumstances, the head may be invaded and replaced by new bone, and the articular cartilage may be either replaced by fibrocartilage or rejuvenated by early reestablishment of an underlying living bone. In this event, a good functional result is obtained, although it may be followed some years later by degenerative arthritis.

In a previously reported case[12] of non-union of two months' standing with death of the head, bony union followed fixation with a Smith-Petersen nail. The head was gradually invaded and replaced, but not before a small portion of the top had collapsed (Fig. 8-A). Function of the hip returned to normal, except for slight limitation of flexion, and has remained so for thirteen and a half years. A recent roentgenogram (Fig. 8-B) shows almost complete reconstitution of the head. The general density is the same as that of the surrounding bone, except for a few persisting areas of radiolucency in the superolateral

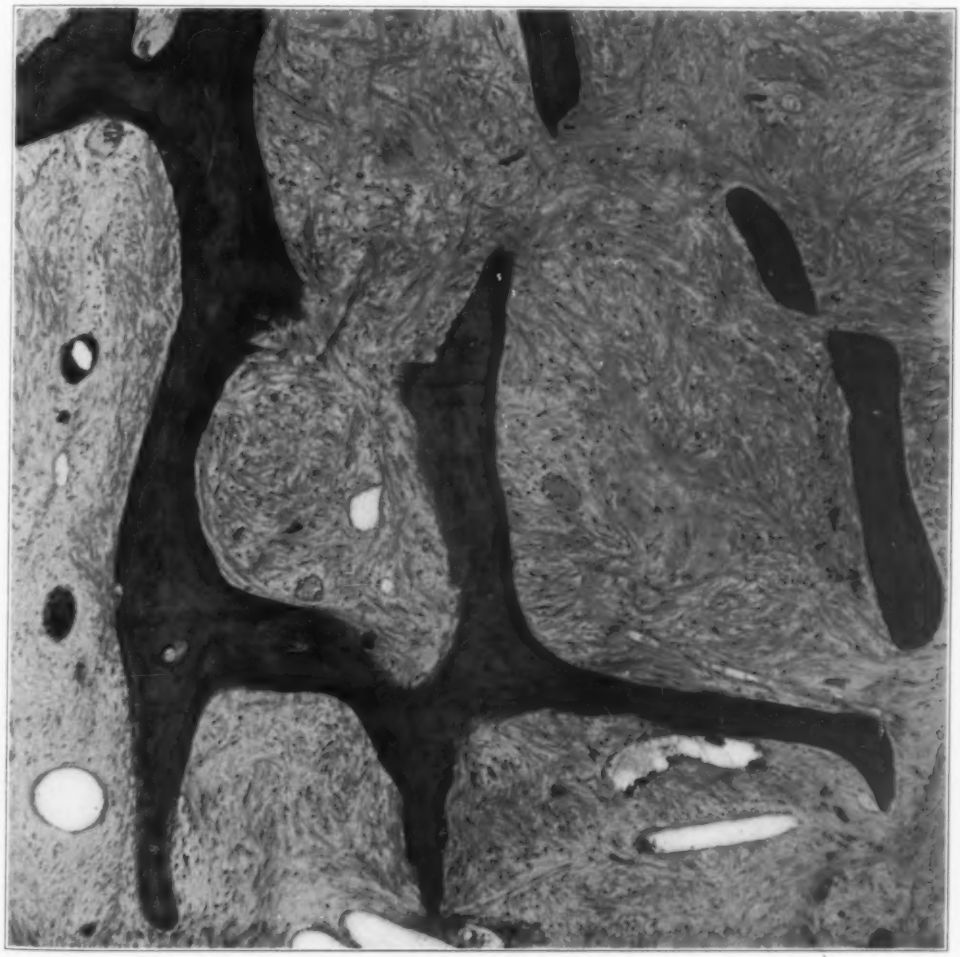

FIG. 6-F

Necrotic trabeculae at center of head, completely surrounded by dense inactive fibrous tissue. There is no sign of new bone being formed.

FIG. 6-G

Lower edge of head, showing many osteoclasts absorbing dead bone, while osteoblasts are laying down new bone on other surfaces.

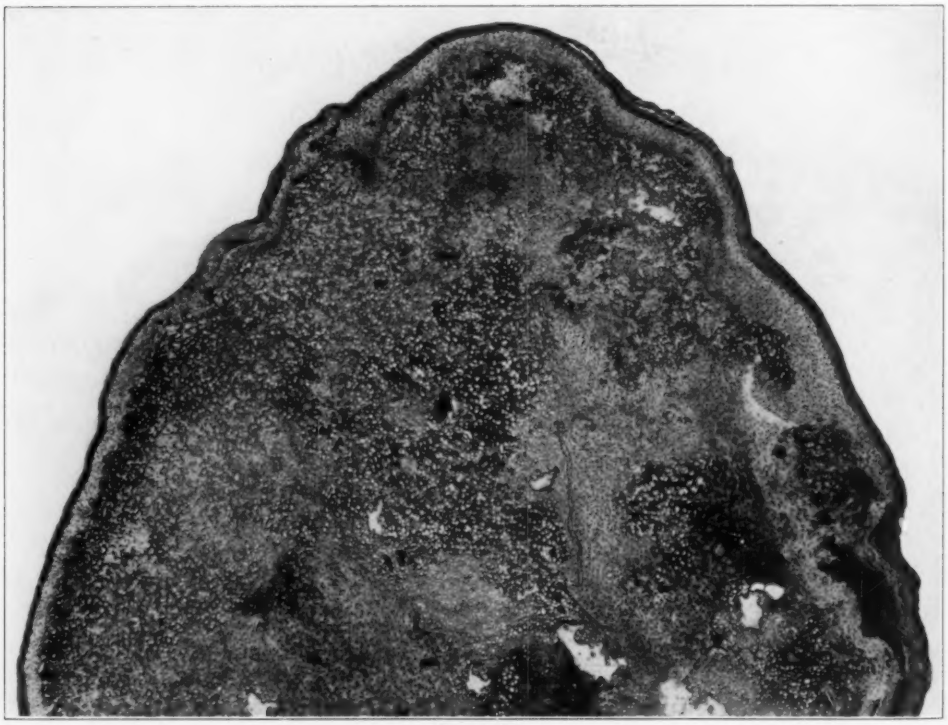

FIG. 6-H

Microscopic section of one of the loose bodies removed from the hip joint.

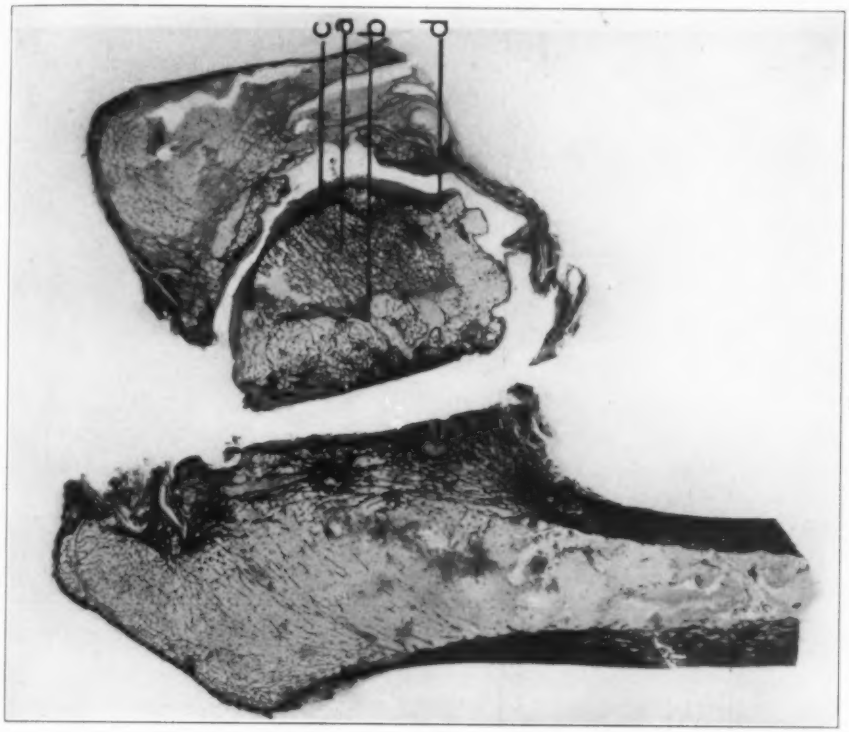

FIG. 7-B

Microscopic section of the hip joint, showing the same changes as Fig. 7-A. Letters refer to photomicrographs.

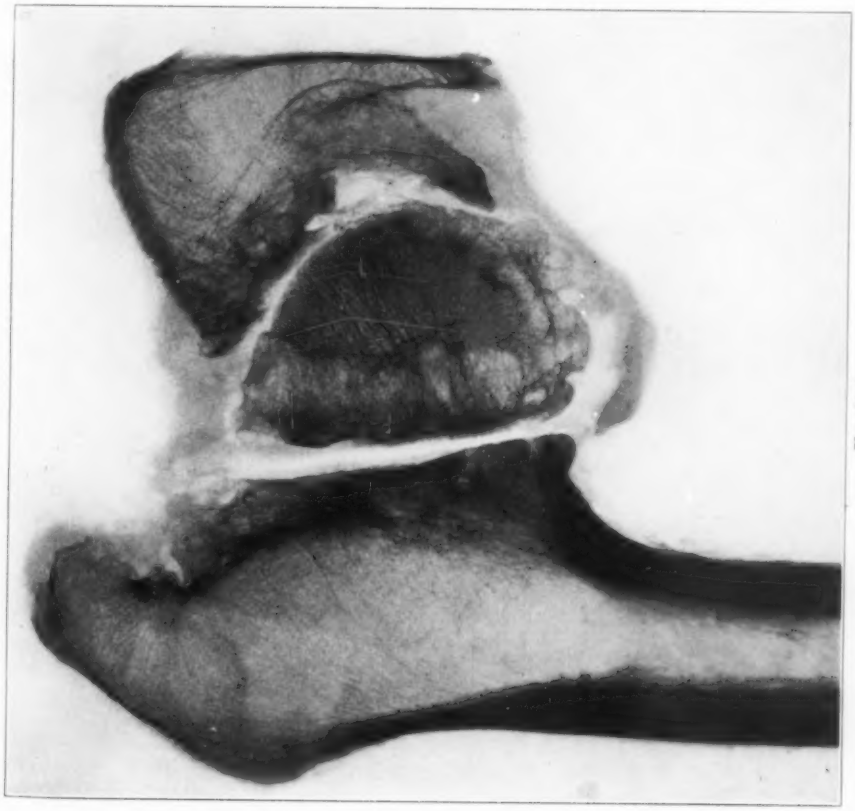

FIG. 7-A

Case 7. Roentgenogram of a slice of the hip joint shows ununited fracture with sclerosis of the margins, complete absorption of the femoral neck, the dense center of the femoral head surrounded by bone of decreased density, and gross degeneration of the joint surfaces.

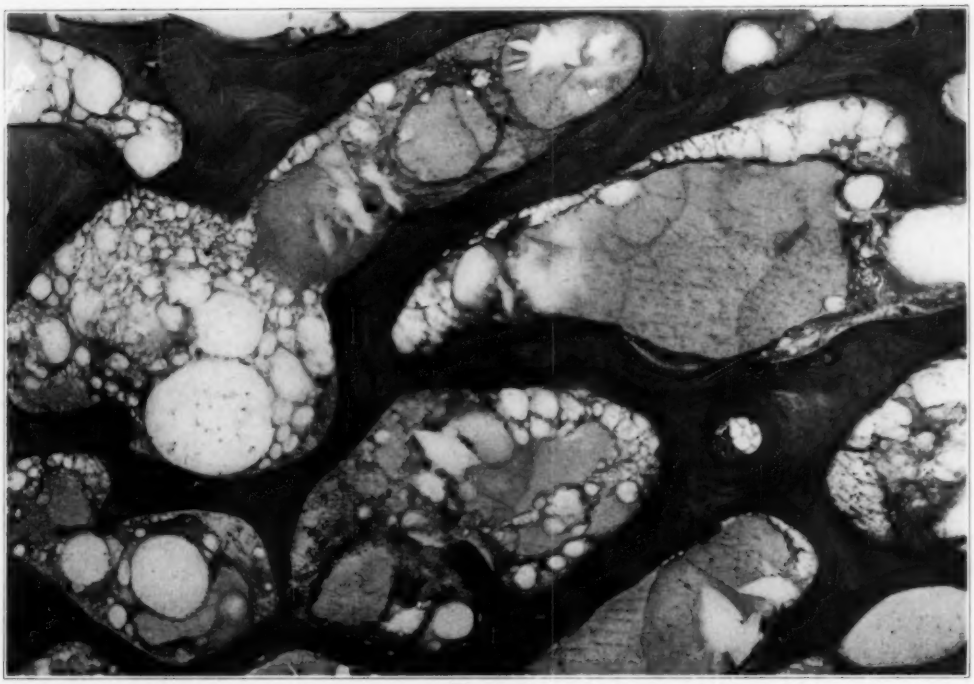

FIG. 7-C

Completely necrotic old bone and marrow in center of dense portion of head.

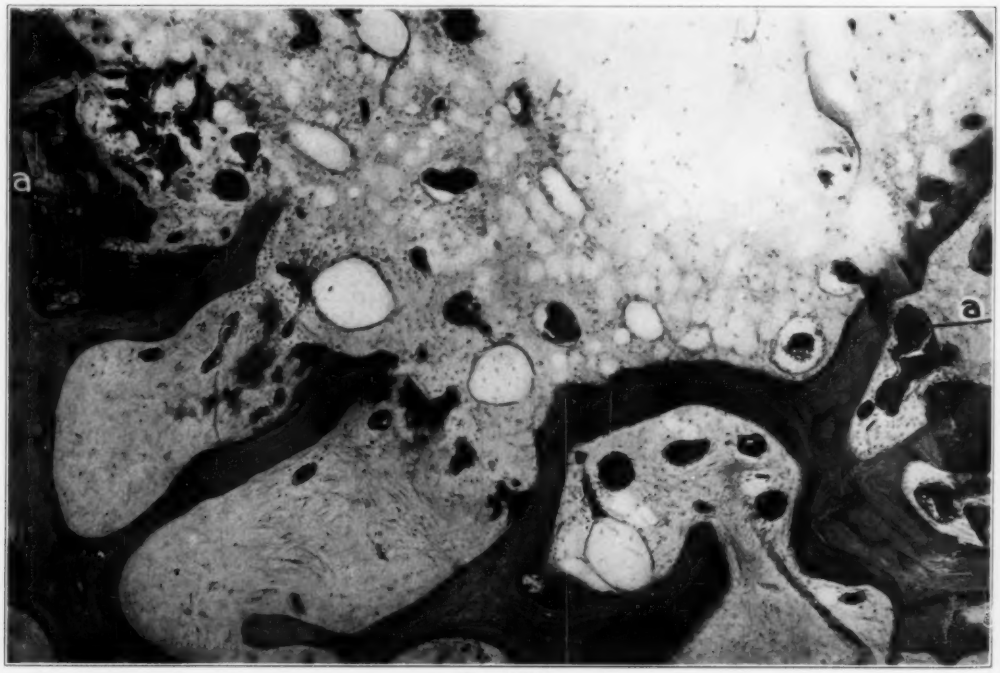

FIG. 7-D

Zone of transformation with new vascular living marrow in upper portion. In the lower portion is dead bone and inactive fibrous tissue, in which are several areas of calcification (*a*).

portion. The contour of the head is slightly irregular and, although the cartilage space is as wide as that of the other hip, there is beginning sclerosis and osteophyte formation on the acetabular side of the hip. Of interest is the presence of marked sclerosis of the opposite sacro-iliac joint.

That a good functional result, lasting for many years, may be obtained in case of a similar fracture of longer standing and in older persons is illustrated by another previously reported case[12], recently restudied.

A woman, fifty-two years old, had an ununited fracture of ten months' standing and a roentgenogram (Fig. 9-A) showed normal even density of the upper portion of the head, while the density of the

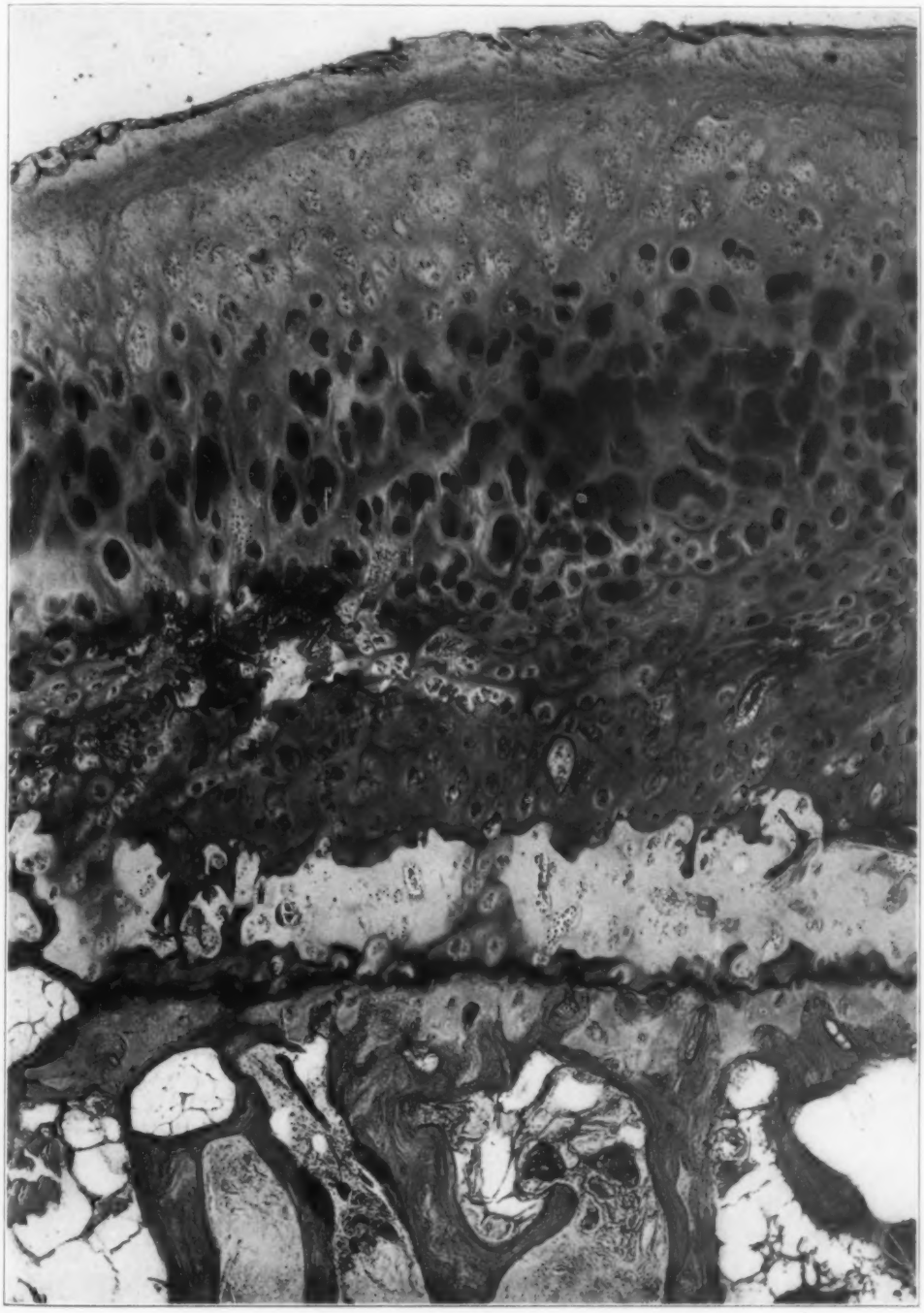

FIG. 7-E

Necrotic bone and marrow at surface of dense center of head. The old necrotic articular cartilage has been invaded by vessels from the foveal area, and calcification and ossification are taking place along them. The surface is overgrown with a new layer of living fibrocartilage.

FIG. 7-F

Section from lower margin of head, where replacement is complete, shows living bone and marrow with revitalized articular cartilage.

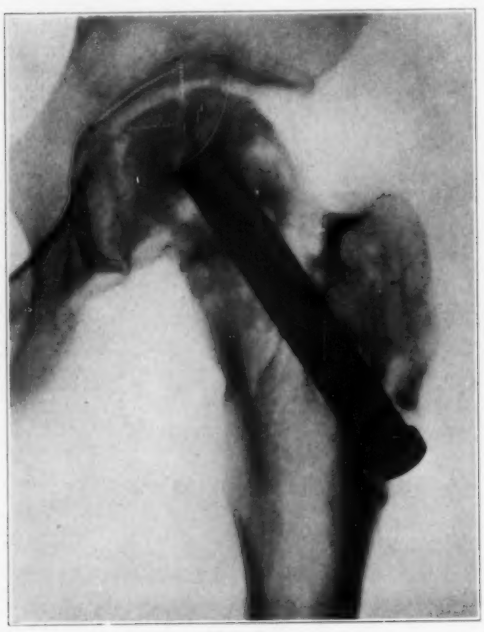

FIG. 8-A

FIG. 8-B

Fig. 8-A: Case 8. One year after fixation of a fracture of the femoral neck, with non-union after

Fig. 8-A: (*continued*) two months. There has been partial replacement of the necrotic head with collapse of a small portion of uninvaded bone at the top.

Fig. 8-B: Thirteen and one-half years after operation. There is complete replacement of the head. The joint space is preserved, but the contour of the head is irregular and there is beginning degenerative arthritis.

FIG. 9-A

FIG. 9-B

Fig. 9-A: Case 9. Fracture in a woman, fifty-two years old. Roentgenogram taken ten months after fracture, showing death of femoral head with absorption and extensive replacement of its lower portion by new bone of low density.

Fig. 9-B: Ten and one-half months after operation, showing extensive replacement of head.

lower portion was greatly reduced and irregular. This was evidence of death of the head, and absorption and replacement of the lower portion by less dense spongy new bone. The fracture was openly reduced and fixed by two tibial bone grafts and three threaded wires. Healing occurred promptly with excellent return of function which has been maintained up to the present, seven and one-half years later. The wires were removed after eight months. The roentgenograhic appearance is shown ten months afterward (Fig. 9-B), and seven and one-half years afterward (Fig. 9-C). There was a high degree of replacement of the upper portion of the head after ten months, and complete replacement without degenerative arthritis or collapse of the head after seven and one-half years (Fig. 9-C). The drilling of the upper portion of the head for bone-grafting may have hastened the invasion and replacement of the dead bone by new bone. The possibility of impairment of function from the development of degenerative arthritis in the future is not precluded.

FIG. 9-C

Dead bone of head has been completely replaced by new bone; the joint changes are minimal.

SUMMARY AND CONCLUSIONS

1. The incidence of non-union is disproportionately high in intracapsular fractures of the neck of the femur, except in those which are either impacted or treated by accurate reduction and internal fixation. Factors entering into this high incidence are the frequent occurrence of death of the head fragment from disruption of the blood vessels of the neck, displacement of fragment ends, poor immobilization, and failure of peripheral callus formation due to absence of a cambium layer on the neck.

2. If the head fragment survives, it undergoes atrophy of disuse of the same degree as that of the distal fragment, and the two fragments are of equal density on the roent-

genogram. Treatment to produce union is usually successful and extensive degenerative changes in the joint do not occur.

3. If the blood supply to the proximal fragment is severed, so that the head dies, disuse atrophy cannot occur, and the head eventually appears on the roentgenogram to be more dense than the surrounding atrophic, living bone. As slow invasion of the head by vascular fibrous tissue occurs, with absorption of the necrotic bone and replacement by cancellous new bone, the roentgenogram shows areas of reduced density.

4. The necrotic articular cartilage remains relatively unaltered until a blood supply reaches it, when it is replaced either by an imperfect type of fibrocartilage or in part by bone.

5. Transformation of a dead head takes place first at the margins of the fracture and at the fovea, and the advancing replacement zone is the weakest part of the bone. Pathological fracture between the dead and living portions may result in collapse and delayed union or non-union.

6. If union has occurred, either primarily or as the result of surgical intervention in the presence of a dead head, and if weight-bearing is begun before replacement is complete, the head will probably collapse, and degenerative changes will be severe on both sides of the joint. If the head is adequately protected, it may be transformed without any collapse; the degenerative changes in the joint are minimal; and a good functional result is obtained.

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Note: Discussion of this paper and of the other papers of this Symposium will appear in the April issue of *The Journal*. *Editor.*

COMPLICATIONS OF OLD FRACTURES OF THE NECK OF THE FEMUR RESULTS OF TREATMENT BY VITALLIUM-MOLD ARTHROPLASTY*

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INTRODUCTION

Vitallium-mold arthroplasty has been performed in forty-two cases, presenting complications of femoral-neck fractures, during the past seven and one-half years. This is a small series, and some of the cases reported fall into the class of "early" rather than "end" results. Our statistics, therefore, may change for better or for worse, as the years go by. It is fair to say that the study of results up to the present time is encouraging.

OPERATIVE PROCEDURES

Mold arthroplasty consists in first creating a joint as nearly perfect, mechanically, as possible, and then guiding nature's repair by means of an inert mold, and carefully supervised exercises. As applied to the treatment of complications of fractures of the femoral neck, the procedure is varied, according to the extent of the degenerative changes; four different types of mold arthroplasty have resulted:

1. Routine mold arthroplasty;
2. Modified Whitman reconstruction operation;
3. Modified Colonna reconstruction operation;
4. Intertrochanteric mold arthroplasty.

FIG. 1-A

Case A. F. Moderate aseptic necrosis of the femoral head in a woman, forty-eight years old. She had sustained a subcapital fracture two years previously and had been treated by traction for five weeks, followed by partial weight-bearing on crutches for six months. Full weight-bearing without pain was possible until sixteen months after the accident; at this time onset of pain necessitated return to crutches. Vitallium-mold arthroplasty was performed two years and three months after injury. This was a very favorable case, because the aseptic necrosis was moderate; bone and muscle atrophy was also moderate, due to the absence of complete immobilization and to persistent, even though limited, function.

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† On Rockefeller Travelling Fellowship.

FIG. 1-B

Two and one-half years after the arthroplasty, the report of the examination read: "Walks without a limp, no pain. One-half inch of shortening. Motions: Hyperextension, 5 degrees; flexion, 115 degrees; internal rotation, 10 to 15 degrees; external rotation, 15 to 20 degrees; abduction, 20 degrees; adduction, 20 degrees." Patient returned to her work as a music teacher in the public schools ten months after the arthroplasty.

This case illustrates the result of a routine mold arthroplasty for limited aseptic necrosis of the femoral head.

FIG. 2-A

FIG. 2-B

Fig. 2-A: A. T. Extensive aseptic necrosis of femoral head and proximal neck in a woman, fifty-seven years old. She had sustained a fracture of the distal neck four and one-half years previously and had been treated by nailing. (This roentgenogram is instructive, because it illustrates the surgeon's habit of outlining the contemplated operative procedure.) In this case the acetabulum was not deepened as planned, because, after removal of the area of necrosis, the neck was found to be too short. Grafts from the iliac crest were introduced into the distal neck and the vitallium mold was applied. The greater trochanter was not transplanted; this was an error in judgment on the part of the surgeon. Six months later the trochanter had to be transplanted, because the reconstructed neck had rotated out of the mold.

Fig. 2-B: Roentgenogram taken eighteen months after arthroplasty, twelve months after transplantation of the greater trochanter. Motions possible at this time: extension, complete; flexion, 90 degrees; abduction, 15 degrees; internal rotation, 15 degrees; external rotation, 10 degrees. There was shortening of five-eighths of an inch. The patient is leading a fairly normal life; swimming and horseback-riding are included in her activities. She uses a cane for outdoor walking.

This case illustrates the result of a modified Whitman arthroplasty for extensive necrosis of the femoral head and proximal neck.

1. *Routine Mold Arthroplasty*

In aseptic necrosis of the head, limited in extent and occurring after union of the fractured neck, a routine mold arthroplasty is indicated. This consists in reshaping the head and acetabulum so as to create two congruous surfaces; the "crater" representing the necrotic area is excised down to bleeding bone and, in cases of relatively extensive necrosis, the resulting defect is packed with cancellous bone from the iliac crest. The reshaped femoral head is covered with a vitallium mold and replaced in the acetabulum (Figs. 1-A and 1-B).

2. *Modified Whitman Reconstruction Operation*

When the aseptic necrosis involves most of the head, even though the fracture has united, a modified Whitman reconstruction is indicated. This consists in discarding the "dead head", reshaping

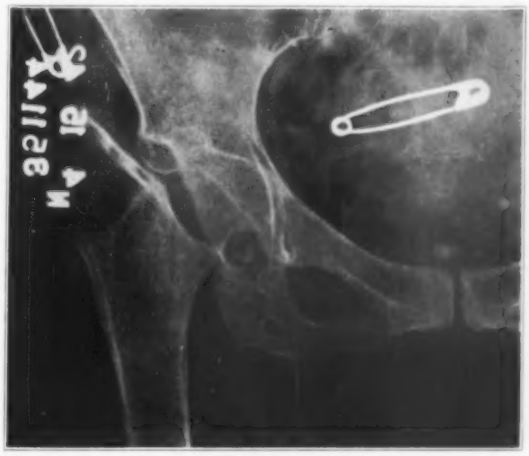

FIG. 3-A

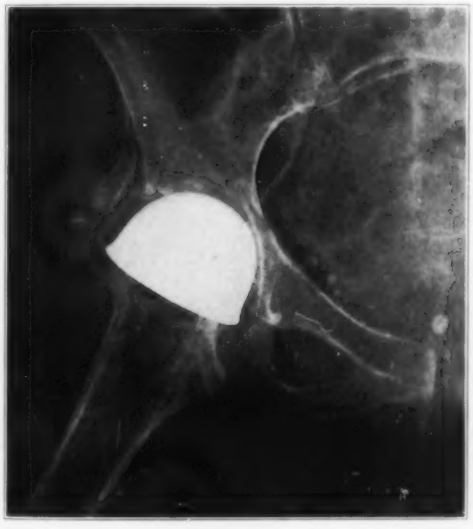

FIG. 3-B

FIG. 3-C

Fig. 3-A: H. P. Complete absorption of right femoral head and neck with marked atrophy of pelvis and shaft of femur in a woman, fifty-eight years old. She had sustained a fracture of the distal neck four and one-half years previously and had been treated by internal fixation by means of two pins. Non-union had become evident six months later and the pins had been removed. Roentgenogram illustrates a local condition most unfavorable for arthrodesis or osteotomy.

Fig. 3-B: One month after a "modified Colonna". The acetabulum has been deepened medially and posteriorly and its anterior wall excised. The anterior third of the ilium has been osteotomized vertically, and its outer cortex displaced laterally; bone grafts from the iliac crest have been packed into the osteotomy gap. In the abducted position, the trochanter, covered by the mold, is in apposition to the floor of the acetabulum. The superior aspect of the lesser trochanter has been excised to prevent impingement against the inferior acetabulum.

Fig. 3-C: Four years after mold arthroplasty. With the hip in neutral, as far as abduction and adduction are concerned, the trochanter rests against the lateral acetabulum; the osteotomy of the ilium has extended the superior acetabular border, thereby preventing subluxation of the femoral head. There is a wide joint line, indicative of a cartilaginous lining of the acetabulum. Motions possible at this time: extension, complete; flexion, 110 degrees; abduction, 25 degrees; adduction, 30 degrees; internal rotation, 25 degrees; external rotation, 25 degrees. There was shortening of two inches. This patient does all her household duties; climbs stairs normally; uses a cane only for outdoor walking; does not complain of pain.

the viable remaining neck, covering it with a vitallium mold, and replacing it in the acetabulum. In the majority of cases, it has been our experience that the viable neck is too short and that osteotomy of the greater trochanter and transplantation to the subtrochanteric region are indicated. This is, therefore, a typical Whitman reconstruction procedure, with the addition of the vitallium-mold arthroplasty (Figs. 2-A and 2-B).

FIG. 4-A

3. *Modified Colonna Reconstruction Operation*

In non-union with a dead head and more or less complete absorption of the neck, a "modified Colonna" operation is indicated. This consists in creating a deep acetabulum and freeing all muscle attachments from the greater trochanter down to the infratrochanteric region. After the trochanter has been shaped with reamers, care being taken to sacrifice a minimum amount of bone, the mold is applied and the greater trochanter is intro-

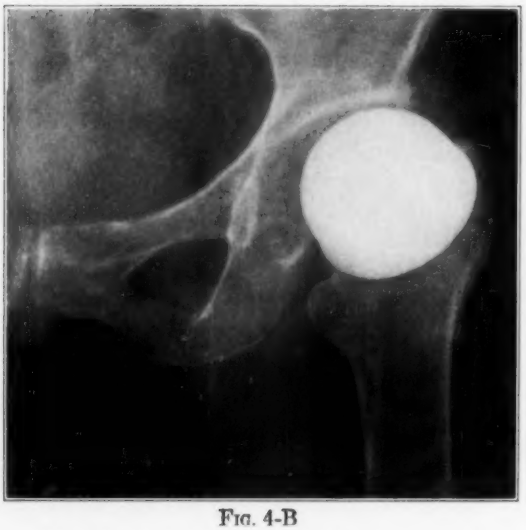

FIG. 4-B

FIG. 4-C

Fig. 4-A: B. G. Aseptic necrosis of femoral head and proximal neck in a woman, forty-nine years old. She had sustained a mid-neck fracture twenty-two months previously, and had been treated by plaster cast for seventeen weeks. Eleven months after the fracture, "nailing" had been done. Fibrous union with extensive aseptic necrosis had resulted.

Fig. 4-B: Eight months after the mold arthroplasty. There was an error of judgment on the part of the surgeon in this case; the greater trochanter should have been osteotomized and transplanted downward, in order to create a better mechanical joint. Aseptic necrosis had progressed inside the mold, until the mold came to rest on the greater and lesser trochanters, producing pressure changes accompanied by pain.

Fig. 4-C: Roentgenogram taken seven months after revision of the arthroplasty. At the time of this operation, the greater trochanter presented such extensive changes that a modified Colonna was considered inadvisable. The lesser trochanter was excised subperiosteally, and the intertrochanteric region was reshaped to fit the mold. The atrophied greater trochanter, with its muscle attachments, was transplanted to the infratrochanteric region. Examination at this time showed mobility as follows: extension, complete; flexion, 95 degrees; abduction, 50 degrees; adduction, 5 degrees; internal rotation, 20 degrees; external rotation, 20 degrees. There was shortening of two inches. The patient does not complain of pain, but has a marked short-leg limp.

This case illustrates the importance of creating a joint mechanically able to transmit weight, even if this requires transplantation of the greater trochanter.

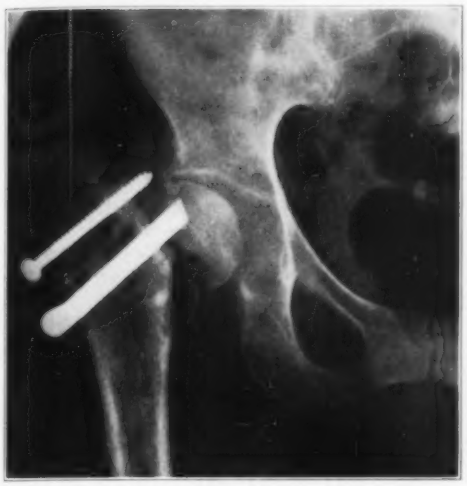

FIG. 5-A

FIG. 5-B

Fig. 5-A: A. M., a woman, fifty-six years old. Showing a "dead head", absorption of the neck, and advanced atrophy of the greater trochanter. There is also evidence of bone absorption around the nail and the screw. In this case, also, the local condition is unfavorable for osteotomy or arthrodesis.

Fig. 5-B: Roentgenogram taken twenty-seven months after a modified Colonna with osteotomy of the greater trochanter, as described in the text. Report of examination: Walks without a cane with some limp; no limp when walking with a cane. Extension, complete; flexion, 115 degrees; abduction, 25 degrees; adduction, 15 degrees; internal rotation, 35 degrees; external rotation, 35 degrees. Patient does not complain of pain. She returned to her work as a nurse, one year after the arthroplasty.

duced into the acetabulum. The divided muscles are transplanted to the infratrochanteric region and fastened by heavy silk sutures or by wire.

Since the greater trochanter inclines laterally from the axis of the femoral shaft, it has a tendency to be displaced outward from the acetabulum when the extremity is brought into a position of adduction. To prevent subluxation, the anterior inferior ilium is osteotomized vertically, and its outer cortex is sprung laterally; bone grafts from the iliac crest are introduced into the cleft, maintaining the outer cortex in its lateral displacement. By this procedure the acetabulum is deepened by half an inch or so. The lesser trochanter sometimes comes into dangerous proximity to the anterior lip of the cotyloid notch, particularly with the extremity in adduction. Subperiosteal excision of the lesser trochanter, with the distal expansion of the iliopsoas attachment left intact, prevents such mechanical impingement.

4. *Intertrochanteric Mold Arthroplasty*

In one patient, extensive degenerative changes occurred under the mold, allowing the mold to settle down and come to rest on both trochanters. The greater trochanter became so atrophied that it could not be shaped and used for a "modified Colonna". After subperiosteal excision of the lesser trochanter, the intertrochanteric region was reconstructed to fit the mold, and the atrophied greater trochanter was transplanted to the infratrochanteric region of the shaft. The result looks good on paper, as far as mobility is concerned, but the shortening is deplorable (Figs. 4-A, 4-B, and 4-C).

In another case the greater trochanter was similarly affected, but for a different reason. This time the changes were due to extravagant use of the means of internal fixation,—namely, a three-flanged nail accompanied by a screw. In this case the initial arthroplasty was successful and, in the opinion of the authors, it is a better procedure than that just described. The screw had already performed a partial osteotomy, and it was simple to make the osteotomy complete after removal of the screw. The osteotomy performed was similar to a green-stick fracture. The remnant of the atrophied greater trochanter was

then folded downward and medially, and transfixed in this position by means of a small three-flanged nail. As has already been pointed out, the greater trochanter deviates laterally from the axis of the femoral shaft; this procedure, then, has a favorable effect on the trochanter, as regards its stability in the acetabulum (Figs. 5-A and 5-B).

EVALUATION OF THE RESULTS OF THESE OPERATIVE PROCEDURES BASED ON PERSONAL EXAMINATIONS AND INTERVIEWS

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The postoperative results in this series of forty-two cases have been assessed by clinical and roentgenographic examinations, and an effort has been made to determine the value of the operation both to the patient and to the surgeon.

Pain in the hip region is minimal in the majority of the cases, and in both early and late postoperative phases. It is usually present in the form of stiffness on rising from a low chair or at the commencement of activity. Patients do not complain of low-back symptoms, nor of stiff knees.

Motion

Mobility of the hip joint has to be considered in conjunction with stability. The greater range of rotation and abduction seen in cases treated by the "modified Colonna" procedure is associated with diminished muscle leverage.

The range of motion achieved by the four operative procedures is shown in Table I.

This motion is invariably smooth and painless, and in no case is there any suggestion of telescoping. It is to be noted that the great majority of these patients have a sufficiently extensive range of hip-joint motion to enable them to dress themselves, including putting on their own shoes and stockings, to sit in comfort in low chairs for protracted periods, and to perform their ordinary household and social activities.

Gait is dependent upon the smooth motion in the hip joint, the stability of the mold in the deepened acetabulum, and muscle power. Strict training and re-education with particular attention to heel-toe movement is most important; and, even in the presence of limb shortening, it is advisable not to raise the heel on the side of the arthroplasty, so that the patient will stretch out the hip and avoid an adductor gait in the early stages. Later, when a good joint is becoming apparent, compensation for shortening is allowable and helps to overcome any Trendelenburg appearance. Unless the gluteal muscles are very weak and the patient is without the support of a cane, a Trendelenburg gait is rarely obvious in these patients, even when climbing stairs.

Residual deformity is more likely to occur, if the operation is complicated by sepsis or failure to maintain the relationship between the mold and the acetabulum. In the majority of the cases, muscle training and exercises prevent any persistent joint deformity, and some degree of shortening alone is present. This is minimal in the patients treated by the routine mold arthroplasty and is more marked after the Colonna and intertrochanteric arthroplasty procedures.

Evaluation of Treatment

The value of this operative treatment to the patient is obvious. Without exception, the younger patients have returned to their work, including such occupations as mining engineer, department-store manager, stenographer, and nurse. The more elderly have been able to perform their social activities, and use a cane for distance walking.

In Table II the functional value of the hip joint, from the point of view of both the patient and the surgeon, is presented; it is to be noted that in two cases the hip is satisfactory to the patient, but not to the surgeon. In three cases, the result is unsatisfactory

TABLE I

RANGE OF MOTION

Hip Function (*Degrees*)	Mold Arthroplasty *20 Cases*	Modified Whitman *5 Cases*	Modified Colonna *14 Cases*	Intertrochanteric Arthroplasty *3 Cases*
Flexion greater than 100	5	–	2	–
between 80 and 100	5	2	3	2
between 60 and 80	7	1	7	–
less than 60	–	2	2	–
Rotation greater than 25	7	1	7	–
between 15 and 25	5	2	4	2
less than 15	5	2	3	–
Abduction–adduction greater than 25	6	1	8	2
between 15 and 25	5	3	3	–
less than 15	6	1	3	–
Flexion deformity 10 to 25	1	3	3	–
less than 10	3	–	3	1
External rotation deformity 10 to 20	–	–	2	–
less than 10	–	1	1	–
Unrecorded	3	–	–	1
Average postoperative shortening	¾ in.	1 in.	1½ in.	2 in.

to both patient and surgeon; and in one case, the patient is dissatisfied, although clinically and roentgenographically the result appears to be good. In the remaining thirty-two cases, both patient and surgeon are satisfied.

Three patients lead a wheel-chair existence due to the following associated illnesses: Parkinson's disease; senility and septic arthritis following nailing; and senility and psychopathic disturbance, with no postoperative rehabilitation for three years.

Complications

Postoperative complications occurred in thirteen patients, five of whom had been operated upon for aseptic necrosis and eight for non-union. They may be grouped as follows:

1. Superficial sepsis with subsequent sinus formation in the iliofemoral scar occurred in two cases. In one case, the infection resolved, but a psychopathic disturbance interfered with postoperative rehabilitation, and the result of the arthroplasty was unsatisfactory. In the second case, revision of the mold arthroplasty and a modified Colonna procedure produced a satisfactory result with resolution of the infection.

2. Deep sepsis was seen in four instances, in two of which the infection originally complicated the nailing operation. One patient died of septicaemia, four months after arthroplasty. In two patients the mold was displaced from the acetabulum; and revision, although unsuccessful in overcoming the subluxation or an occasionally draining sinus,

TABLE II

FOLLOW-UP STUDY

PROGRESSIVE IMPROVEMENT AND ESTIMATION OF VALUE OF OPERATION

Years after Operation	Average Flexion Range (*Degrees*)	Condition of Hip			
		Satisfactory (*No. of Cases*)		Unsatisfactory (*No. of Cases*)	
		To Patient	To Surgeon	To Patient	To Surgeon
0–2	79	16	17	1	–
2–3	81	3	2	1	2
3–4	85	3	3	1	1
4–5	90	6	5	–	1
Over 5	86	6	5	1	2

enabled satisfactory function to be regained. In the fourth case, septic arthritis had complicated the original nailing operation, and a modified Colonna procedure was done one month after drainage of the joint and removal of the femoral head. Good healing and function were obtained; but, fifteen months later, a blow on the hip resulted in formation of a small sinus, and revision may be necessary if osteitis under the mold and sinus formation persist.

3. Subluxation of the mold upward and laterally occurred in three cases, two of which were treated by the modified Colonna revision and the third by a modified Whitman procedure.

4. Avascular necrosis within the mold was seen in one case, and was apparent clinically by persistent pain and muscle spasm. Revision by intertrochanteric arthroplasty produced a good result.

5. Hypertrophic changes at the mold margin and a flexion-adduction-external-rotation deformity was seen in two cases, one of which was successfully treated by a modified Whitman procedure; in the other case the mold was removed at another hospital, and a subtrochanteric fracture, incurred during the operation, was used as an osteotomy. A satisfactory result is reported, but is not included in this series.

6. Upward displacement of the greater trochanter, in spite of fixation to the femoral shaft by a three-flanged nail, occurred in one case. Weak abductor muscles resulted, but the patient walked well with a cane.

7. Pulmonary embolism did not occur in this series of cases, and prophylactic femoral-vein ligation was used in only one case.

Mortality

There was no operative mortality. One patient died from septicaemia, the source of which was septic arthritis of the hip following arthroplasty four months previously; and two other patients died of carcinoma of the stomach and coronary occlusion, respectively, two and one-half and one and one-quarter years after operation. It is to be noted that half of the patients in this series of cases were sixty years of age or more.

SUMMARY AND CONCLUSIONS

The results of treatment of complications of fractures of the neck of the femur by vitallium-mold arthroplasty have been studied in forty-two cases. Five cases have been chosen as instructive and illustrating the various operative procedures employed.

Based upon this study the following conclusions have been drawn:

1. Eighty-five per cent. of the results are satisfactory to both patient and surgeon.

2. There is progressive improvement in function for three to four years after operation. The condition then becomes stationary. (The postoperative period in the oldest case is now seven and one-half years.)

3. In this series of patients, low-back symptoms, stiff knees, or postural difficulties were not present.

NOTE: Discussion of this paper and of the other papers of this Symposium will appear in the April issue of *The Journal*.

Editor

PAINFUL, NON-SUPPURATIVE, LOCALIZED SCLEROSIS OF THE LONG BONES

With a Report of Two Cases

BY WILLIAM MACKENZIE, F.R.C.S. (EDIN.), NEWCASTLE-UPON-TYNE, ENGLAND

Sclerosing non-suppurative osteomyelitis was described by Garrè in 1891, before the roentgen ray was available. Jaffe and Lichtenstein point out that the translated title of Garrè's original paper is "Some Unusual Expressions of Acute Infectious Osteomyelitis", and that one form of these "expressions" is sclerosing non-suppurative osteomyelitis, which was described as setting in acutely with pyrexia, local pain, and swelling. The temperature and soft-tissue swelling subsided; osseous enlargement persisted. This condition has received roentgenographic recognition, and is thus described by Gray:

> "*Sclerosing Osteomyelitis* (Garrè): This is a rare form of chronic non-suppurative osteomyelitis, generally affecting either the tibia or femur in older children and young adults. It is characterised by a diffuse area of sclerosis near the end of the diaphysis, gradually fading off into normal bone.
>
> "The cortex is increased in extent and density, and the medullary cavity may be so much encroached upon as to be unrecognisable. A somewhat fusiform expansion of the shaft results, and there is generally slight roughening of the surface. The X-ray appearances in the early stages of some forms of *Ewing's* tumour may be indistinguishable from this condition."

Some French writers describe cases of osteomyelitis which are "chronic from the beginning" (*chronique d'emblée*). Laffaille records two such cases *à forme pseudo-tumorale*.

Fosdick Jones recorded the case of a boy whose tibia was affected. In June 1918, the patient sustained an injury to the shin, which left no residual discomfort. In December 1918, he sustained another injury at the same site; pain and pyrexia immediately followed and persisted. The boy lost weight and strength. When seen in July 1919, he was thin and anaemic; his temperature was 101 degrees; and the tibia showed a well-marked forward bulge. At operation no pus was found, and cultures from the medullary canal were sterile. Microscopically, there were no foci of round-cell infiltration. Fosdick Jones discussed the differential diagnosis from syphilitic periostitis, sarcoma, and "the rare type of solid osteitis fibrosa encountered in the long bones". He considered this type of bone sclerosis to be rare; and, in the discussion which followed the presentation of this case, Ryerson stated that he had never seen a case which could be classified as Garrè's osteomyelitis. On that occasion Sir Robert Jones instanced a case in which there had been recurrence eighteen months after operation. On the authority of Mauclaire, Fosdick Jones quoted Kocher's opinion that a considerable number of bone enlargements, diagnosed as sarcoma and treated with apparent success by amputation, were merely instances of sclerosing non-suppurative osteomyelitis of the long bones.

In 1920 Bloodgood, under the heading of Pyogenic Ossifying Periostitis, discussed a case previously diagnosed as sarcoma of the femur. The patient had refused amputation. Bloodgood commented that: "A case of this kind demonstrates that some of the so-called cures of periosteal sarcoma after amputation, serum, x-ray or radium belong to this type of periosteal lesion". Bloodgood subsequently recognized a resemblance between this case of femoral ossifying periostitis and the case of tibial osteosclerosis described in the communication of Fosdick Jones, in view of which he concluded that his case also corresponded to the non-suppurative osteomyelitis of Garrè. He further stated: "That I have observed seven cases of this type since February, 1921, a period of thirteen months, is an indication that it is a bone lesion which we must bear in mind". Bloodgood attached the name of infectious ossifying periostitis to this condition, and attributed it to metastasis from some focus of infection.

In reviewing a series of thirty cases of Garrè's osteitis seen at the Mayo Clinic, Henderson stated that seventeen patients were operated upon; of these, nine obtained relief;

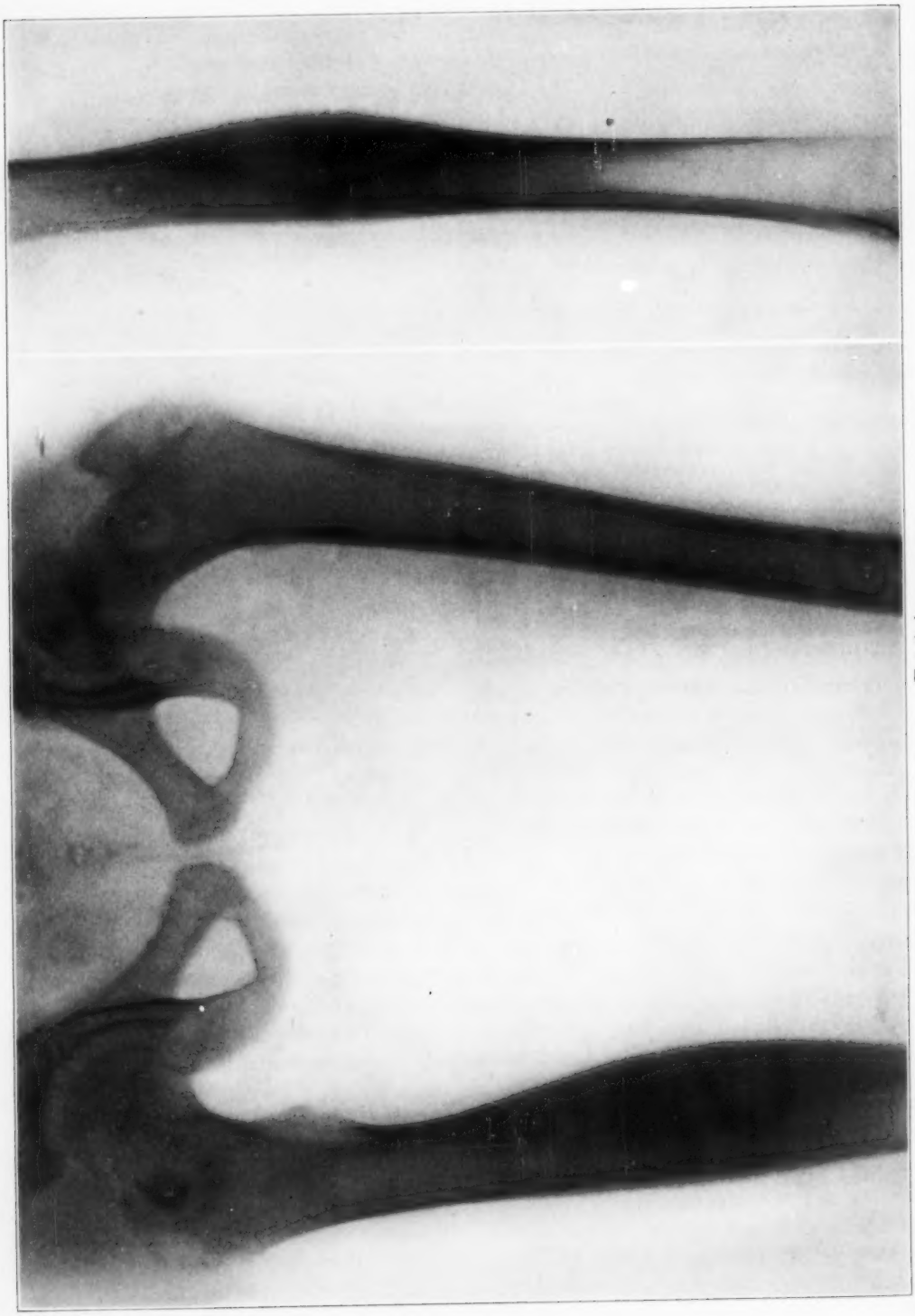

FIG. 1-A

Case 1. March 1938. Anteroposterior and lateral views of affected femur. (Roentgenogram has been reversed.)

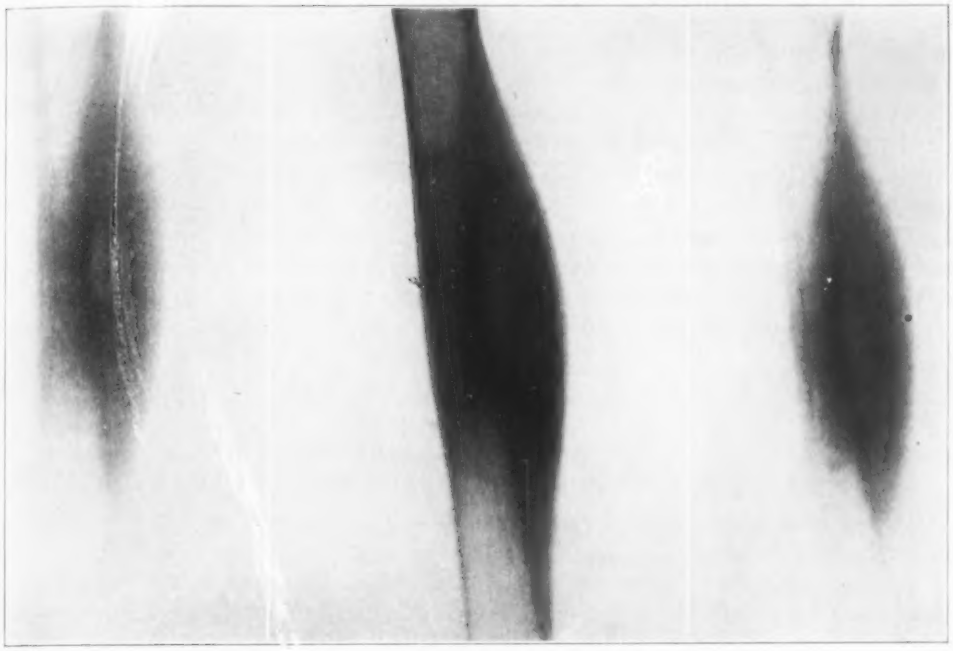

FIG. 1-B

May 1938. Three roentgenograms of varying densities. Illustrations on the right and the left, respectively, indicate the less dense central area.

FIG. 1-A

Case 1. March 1938. Anteroposterior and lateral views of affected femur. (Roentgenogram has been reversed.)

one was not relieved; two had temporary relief; and five were not traced. Thirteen were not operated upon. Of these, three were relieved permanently; one was relieved but had recurrence; and nine were not traced. The surgical procedure adopted was either "guttering" the bone or drilling multiple holes through both cortices. Out of the thirty cases, he states: "In eight, either previous or subsequent to our examination, there was suppuration, but in only one case a history of necrosis of bone". Henderson states that, although onset may be acute and sudden, more often it is subacute and chronic. There may be atrophy of the limb. Tenderness is usually complained of on deep pressure. "The course of the disease is chronic and long. Recurrences, even after relief has been afforded by operation, are not infrequent . . ." In Henderson's series, different types would seem to have been included.

The silent foci of localized osteomyelitis, which are described by Phemister and which are delayed sequelae of acute osteomyelitis, are essentially different, both roentgenographically and clinically, from sclerosing non-suppurative osteomyelitis.

Wishner described five cases of chronic sclerosing osteomyelitis. In his cases, the evidence for chronic bone infection was relatively slight. In a brief review of this subject, Ewing says: "It may well be that the term osteomyelitis is a misnomer . . . While some believe it is due to infectious organisms in the haversian

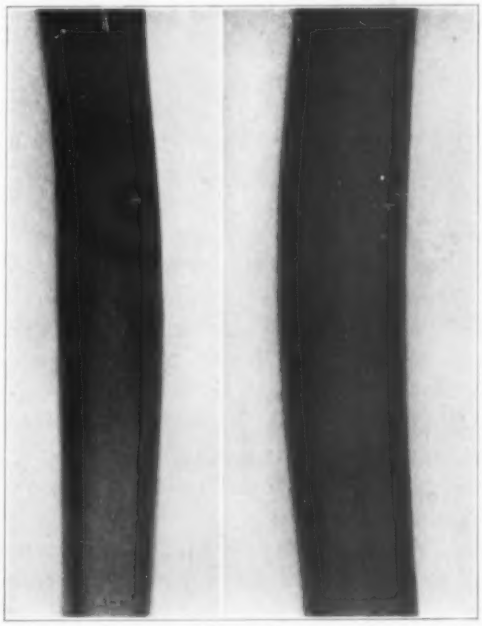

FIG. 1-C

April 1941. Anteroposterior and lateral views of left femoral shaft show disappearance of fusiform enlargement.

spaces which are so attenuated in character as to cause this unusual syndrome, it is more likely that the pathology is due to a diminution in the blood supply locally from some non-infectious irritant, *viz.* trauma."

In 1935, Jaffe described five cases of what he regarded as a benign neoplasm arising within the medulla in spongy bone areas. To this growth he gave the name of "osteoid-osteoma". In 1940 he and Lichtenstein described a further series of such cases, of which thirteen involved the shaft cortex of long bones. The patients were adolescents or young adults. The principal complaint was pain, and "it was this that consistently led the patients to seek medical attention . . . In no case was there a history of febrile episodes in connection with the lesion." Local heat and redness were absent.

Jaffe and Lichtenstein drew attention to two features in roentgenographic appearance of these lesions:

1. The focus (osteoid osteoma proper) is generally indicated by an oval, relatively "clear" area.

2. The surrounding osseous reaction incited in the adjacent bone, when long-bone cortex is involved, results in very marked cortical thickening, which "may extend for several inches both above and below the osteoid-osteoma proper" and may partially envelop the shaft of the bone.

These authors state: "It seems very clear to us that a great many cases which, in the past, have been interpreted specifically as 'osteomyelitis with annular sequestrum', 'chronic bone abscess', 'osteomyelitis with cortical-bone abscess', 'sclerosing non-suppurative osteomyelitis', 'osteomyelitis chronic from the beginning', etc., actually belong in the category of osteoid-osteoma." In their cases, surgical treatment resulted in clinical cure "with prompt and often dramatic relief of distressing pain"; there were no recurrences in seven years.

The clinical and roentgenographic interpretation of this type of bone lesion, put forward by Jaffe and Lichtenstein, has been supported by Harmon, Kleinberg, Lewis, Horwitz, and Stauffer.

Brailsford describes three cases of cortical thickening of the tibia, in which evidence of an inflammatory origin was found on exploration of the bone. (In two cases a "bead of pus" was discovered, and in one of these recurrence took place after operation; in the third case a small cavity suggesting a chronic bone abscess was found, and in this case healing was very slow.) One of his roentgenograms shows persisting tibial enlargement, four years after surgical intervention. With regard to differentiation and acceptance of the bone lesion designated as osteoid osteoma, Brailsford says: "Though the histological appearances as illustrated and described by Jaffe and Lichtenstein lend support to their suggestion that it is a benign tumour of bone, there is nothing in the radiographic appearance which supports this diagnosis."

The presence of visible pus or septic cavitation—even though minute—is not characteristic of the osseous lesion known as Garrè's osteitis. It would appear, therefore, that non-suppurative sclerosis of the cortical bone, accompanied by pain, is a clinical and pathological entity *sui generis*, for the explanation of which the associated existence of a benign neoplasm, designated "osteoid-osteoma", has been postulated by Jaffe and Lichtenstein, who further claim that the lesion admits of roentgenographic verification.

A completely satisfactory interpretation of the total lesion is, however, not provided by the conception of reactive osteosclerosis in relation to a neoplastic center, such as is described by Jaffe and Lichtenstein; these authors, indeed, admit that "why an osteoid-osteoma should arouse a perifocal osteosclerosis is by no means clear".

CASE REPORTS

These cases were characterized by gradual onset and by gradual increase of pain, without an acute phase of illness.

CASE 1. A girl, ten years and ten months of age, who had previously been healthy and without history of injury, was first seen on February 25, 1938. She complained of pain in the left knee and leg, which had begun about three months before; recently it had been severe at night, interfering with sleep.

On physical examination, the child walked with a slight limp (this was said to have been present intermittently for about six months). The femoral shaft was palpably enlarged, and there was tenderness at the level of the center of the enlargement. There was slight wasting, but no apparent affection of the soft tissues or skin; there was no pyrexia; movements were unrestricted.

On roentgenographic examination (Fig. 1-A) the medial aspect of the left femoral shaft showed a dense enlargement, which, at the mid-point, partly obscured the medulla; from that level the thickening gradually diminished, both upward and downward. The cortical thickening extended as high as the lesser trochanter, but did not reach the medial condyle of the femur. In the lateral view, the femoral shaft showed a dense fusiform enlargement; the anterior aspect had a rough "knobby" contour and the posterior aspect had a smooth "layered" appearance.

On blood examination, the leukocyte count was 9550, with 58 per cent. polymorphonuclear cells. A negative Wassermann was found; after "provocative" injections of arsenicals, repeated Wassermann tests were negative. Potassium iodide was administered without effect. There was some oscillation of temperature during this observation period, but later this ceased. Aching in the limb persisted. Further roentgenographic examination, with variation of technique, disclosed what appeared to be a long oval cavity within the thickened and sclerosed bone; the presence of a small sequestrum was faintly suggested (Fig. 1-B).

On June 11, 1938, the bone enlargement was explored and was deeply guttered. Neither pus, nor cavity, nor evidence of sequestrum formation was found. Bacteriological examination of the bone fragments was negative. Histological examination showed dense laminated bone, separated by a cellular connective tissue without inflammatory cell infiltration.

The operative wound healed without inflammatory reaction. Pain in the limb ceased about forty-eight hours after surgical intervention. Tenderness was absent a month later. The child was kept in the hospital until the femoral enlargement was evidently subsiding. When discharged from the hospital on March 11, 1939, she could walk and run well. There has been no recurrence of pain or disability. Roentgenograms of the left femur were taken in 1941; the fusiform enlargement had disappeared, and a barely recognizable trace of the guttering of the bone remained (Fig. 1-C). The left femoral shaft was more massive than the right and showed a slight curvation medialward.

In 1940, the author saw Campbell's roentgenograms of Garrè's osteomyelitis, read his comment on the condition, and noted in particular the negative results of bacteriological examination of this bone lesion. The monograph of Jaffe and Lichtenstein on osteoid osteoma also came to notice, and as a result the symptoms, the roentgenographic picture with cortical thickening and a radiolucent area, and the other findings in Case 1 were recalled.

In May 1945, the following case was seen.

CASE 2. The patient was a boy of fourteen years who, for about two months, had been complaining of aching in the shin. This discomfort was associated with a sensitive "lump" which the boy attributed to a contusion sustained two months before the onset of the pain (although he was uncertain which shin had been injured).

On examination, a forward bulge of the shin border was evident; there was no redness of the skin nor any swelling of the soft tissues. Later there was slight firm thickening without "pittable" oedema. Tenderness, which was acute, was limited to a small area (Fig. 2-A). The pain and the tibial enlargement gradually increased. Roentgenographic examination in July 1945 (Figs. 2-B and 2-B') was considered to justify no more than a diagnosis of chronic osteitis. In September 1945, another roentgenographic examination was made (Figs. 2-C and 2-C'). At that time the cortical sclerosis had increased; in the lateral view a radiolucent area was observed. The roentgenologist (Dr. Donald Ramage) expressed his opinion that the diagnosis of osteoid osteoma was now established. Attention is drawn to a special feature of the "clear" area in the roentgenogram,—namely, that it is not sharply outlined (so also in the previous case).

Pain persisted, increased, and interfered with sleep; assent to surgical treatment was given.

The leukocyte count was 6200, with 53 per cent. polymorphonuclear

FIG. 2-A

Case 2. Visible tibial swelling. Area of maximum tenderness is marked on skin; this site was shown roentgenographically to correspond approximately with that of the tibial focus.

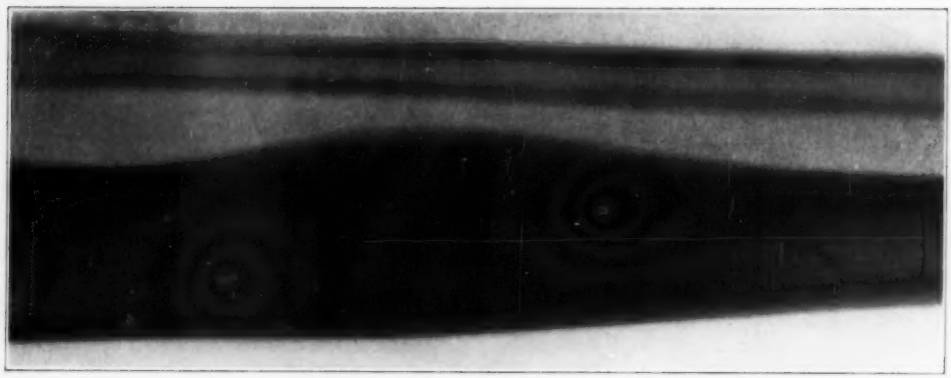

FIG. 2-E
February 28, 1946

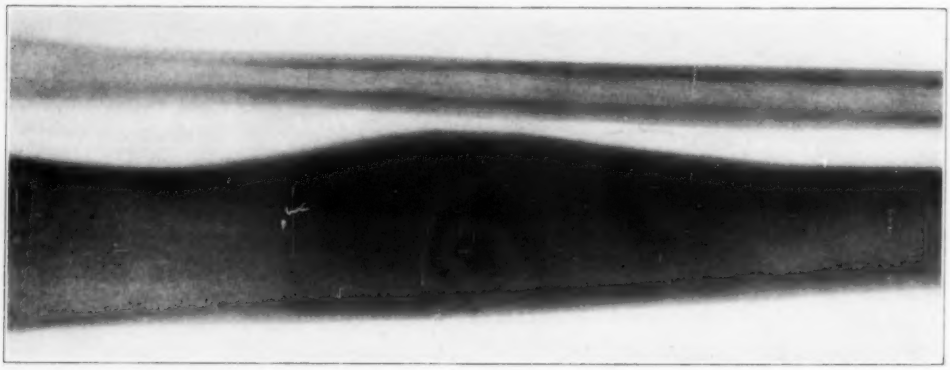

FIG. 2-D
January 23, 1946

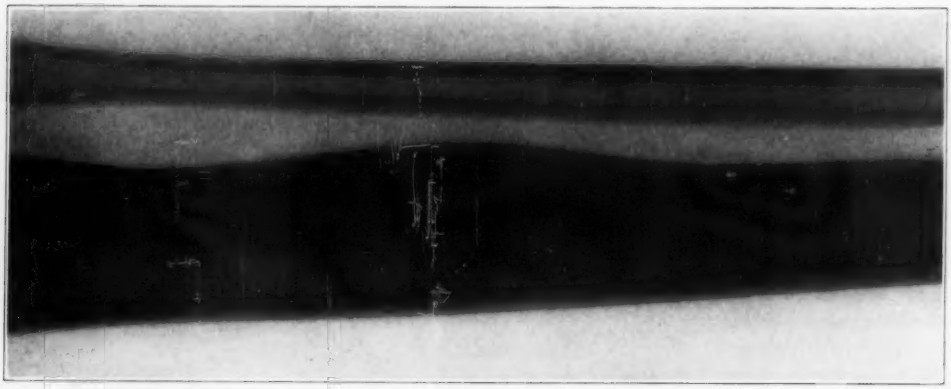

FIG. 2-C
September 26, 1945

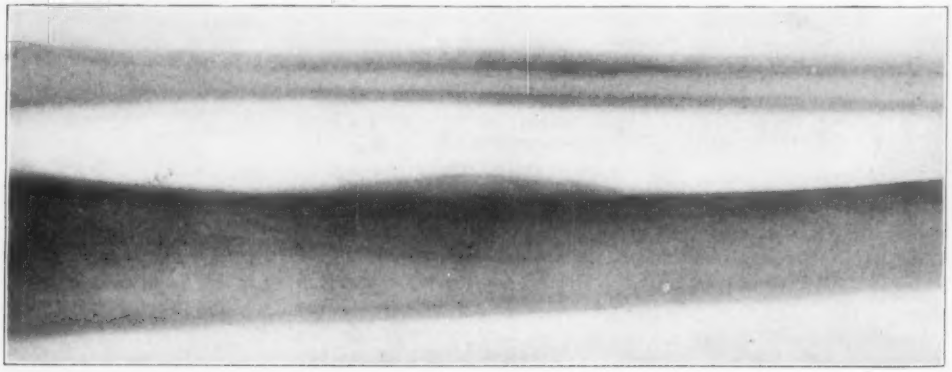

FIG. 2-B
July 9, 1945

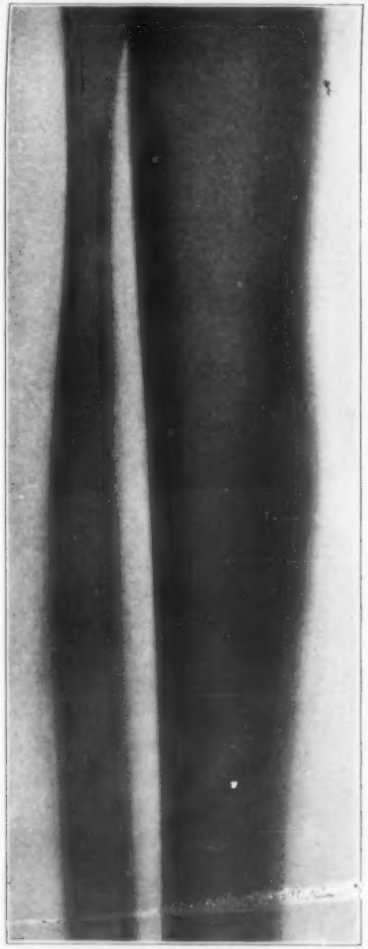

FIG. 2-B'
July 9, 1945

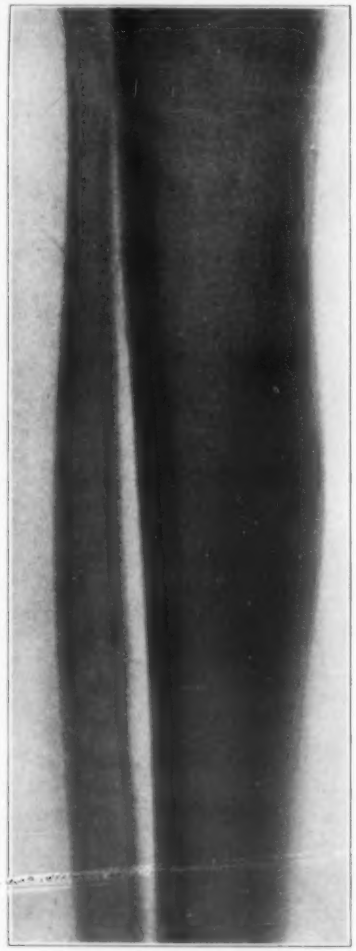

FIG. 2-C'
September 26, 1945

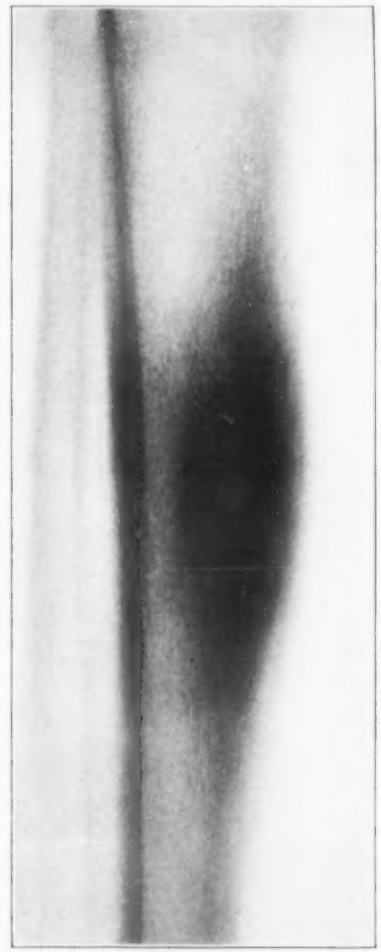

FIG. 2-D'
January 23, 1946

FIG. 2-E'
February 28, 1946

Development of cortical thickening (anteroposterior and lateral views). Note relatively "clear" areas of focus in Figs. 2-D' and 2-E'.

cells. The blood sedimentation rate showed a doubtful increase. The Wassermann reaction was negative on two occasions.

On March 1, 1946, a thick boat-shaped block of bone was removed from the tibial enlargement. The bone was extremely hard. On the deep surface of the exsected portion (which was divided across), a reddish wartlike excrescence was observed (Fig. 2-F). Swabbings from the bone surface laid bare by the exsection and from the deep surface of the exsected bone were sterile; bone shavings from the "saucerized" tibia produced a growth of a diphtheroid organism and a coagulase-negative *Staphylococcus albus* (both regarded by the bacteriologist as contaminants).

FIG. 2-F
Exsected tissue.

The wound healed quickly. Pain ceased two or three days after the operation. At the end of May, the boy walked

FIG. 2-G
Dense portion, low-power photomicrograph.

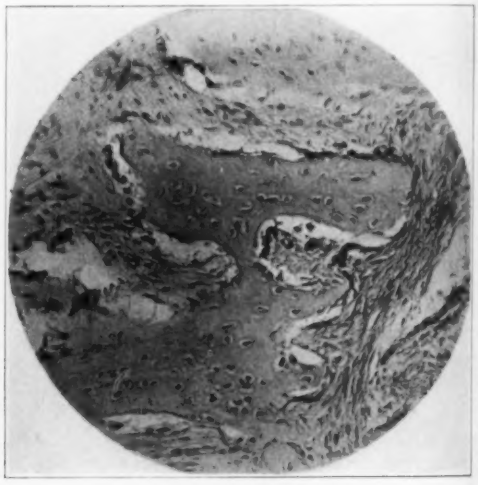

FIG. 2-H
Dense portion, high-power photomicrograph.

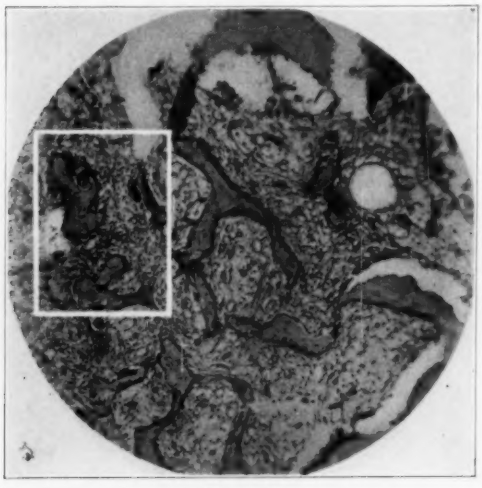

FIG. 2-I
Low-power photomicrograph of nidus. Rectangle indicates area selected for high-power photomicrograph.

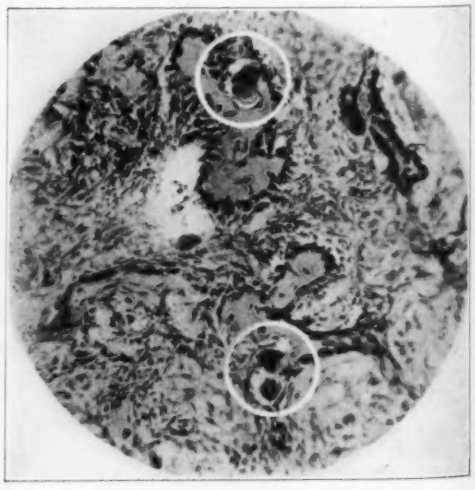

FIG. 2-J
High-power photomicrograph of nidus. Multinucleated giant cells are shown in circled areas.

normally and admitted no disability. There was slight tenderness on firm pressure at the site of the bone exsection. Anteroposterior roentgenograms of the tibia at that date showed sclerosis and broadening; on the lateral view the deep excavation in the sclerosed and thickened bone was evident, but seemed less than it had been in March. The wartlike excrescence, mentioned previously, resembled the tissue described and illustrated by Jaffe and Lichtenstein (in their Case 3).

The exsected bone was examined histologically by Colonel W. F. Harvey, who reported as follows:

"The material was in two halves and consisted of dense, hard bone. One half showed a grooved central space, which contained a noticeable soft mass. This portion of the specimen was selected for section. Peripherally, the section [Figs. 2-G, 2-H, 2-I, and 2-J] shows fibrous tissue and periosteum, the fibrous tissue being loose and vascular and interspersed with fatty tissue, while the osteogenic periosteum is of the usual type,—cellular, fairly compact, and axially layered by osteoblasts.

"Under the periosteum there is a narrow layer of well-differentiated cancellous bone, the cancelli of which are wide, lined by osteoblasts, and contain, not bone marrow cells, but an oedematous-looking reticular-cell tissue which is probably the usual fibrofatty tissue of the marrow. This tissue is vascular—mainly capillary—and the vessels are turgid with blood. The dense cortical bone is succeeded by a central focus, which is approached by loose cancellous bone. The 'nidus' lesion is hemorrhagic and, in the section, is only a comparatively small area of tissue. Bone here is represented by an osteoid, trabeculate meshwork. The trabeculae are apparently osseomucinous (or chondromucinous), and the mucinous areas are lined by active osteoblasts. Moreover, the interstitial matrix to this immature bone is cellular, fibroblastic, and fasciculate. Multinucleated osteoclast cells are, in moderate numbers, easily recognized, but do not suggest osteoclastoma. The histological appearance of matrix, bone, and cells should rather be described as osteoclastoma-like, and as reminiscent of the areas sometimes found in association with osteitis fibrosa cystica (general and localized) and with single bone cysts. There is no microscopic indication of any inflammatory or septic reaction. Syphilis is in no way suggested by any endarteritis, necrosis, or plasma-cell abundance. Tubercle is, histologically, not in question."

SUMMARY

A special feature of these two cases of painful non-suppurative, non-inflammatory cortical thickening of the long bones is that the radiolucent area is not sharply outlined; in this it resembles the illustrative roentgenograms of Jaffe and Lichtenstein, and of others.

On microscopic examination, the bone focus removed in the second case resembled the histological description given by Jaffe and Lichtenstein.

No positive opinion is expressed regarding the essential nature and cause of the bone lesion in the two cases.

NOTE: Grateful acknowledgment is made to Dr. R. W. S. Harvey for the preparation of the photomicrographs.

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THE NEWER PATHOLOGICAL AND PHYSIOLOGICAL CONCEPTS OF ANTERIOR POLIOMYELITIS AND THEIR CLINICAL INTERPRETATION *

BY ARTHUR STEINDLER, M.D., IOWA CITY, IOWA

It has become increasingly difficult to coordinate the great number of observations and studies which have been made on infantile paralysis, especially under the stimulus of The National Foundation for Infantile Paralysis, Inc. The clinical side has remained fairly well standardized for several decades; little that is essential has been added since Lovett and Lucas and Browning elaborated the clinical pathology. The interpretation of the clinical features of this disease, however, has been greatly enhanced by the newer studies in the fields of pathology and physiopathology.

PATHOLOGICAL STUDIES

In the field of pathology, the developments have been slow and laborious. The site of the lesion had been placed by Heine, in his later publications, as the spinal cord; but it was Charcot and Joffroy who recognized the anterior-horn cell as the principal seat of the pathological changes, even though they considered these secondary to interstitial changes. They also described, in autopsies, changes in the anterolateral white fasciculi of the cervical and lumbar regions. From then on the anterior-horn cells held the center of interest, and this was corroborated by many of the earlier autopsies, including those of Taylor and of Edes. The clinician had been depending entirely upon changes in the anterior-horn cells for his interpretation; it was on these grounds that the recession and intermission of the disease, remarkable and perplexing as it was, had been explained. The concept of the so-called "stunned" cell and of transitory oedema was thoroughly exploited.

That the disease is not limited to the anterior-horn cells has been known since 1908, when Cadwalader reported interstitial changes. He held, then, that the changes which terminate in the neuronophagia of the ganglion cells are primary. Lewis, in 1910, reported perivascular infiltration along the vessels in the gray matter, and involvement of the lateral and posterior horns in monkeys; he also found involvement of the white matter, the spinal-root ganglia, and the pia and arachnoid.

At the same time Sachs, who believed, as did Charcot forty years before, in primary interstitial and mononuclear infiltration, pointed out in his autopsies on the human that the changes were not limited to the anterior horn. He specifically called attention to changes in the "central gray matter", probably meaning what we now call internuncial pools; and he likewise found the posterior horns involved.

The picture was further clarified by reports of Jonnesco, who, in two autopsies, found the cells of the spinal ganglion involved in territories corresponding to the paralyzed regions.

Then came the contributions which pointed to the extension of the pathological process beyond the spinal cord. As early as 1910, Krause, who still believed in the secondary involvement of the ganglion cells, described inflammatory foci in the medulla, the pons, and the basal ganglia. A few years later Blanton confirmed in his fifteen autopsies the finding of changes in the basal ganglia and pons, but in twelve cases examined he found the cortex and the cerebellum normal. In fact, none of the subsequent reports on pathological findings failed to include structures of the cord other than the anterior-horn cells, as well as other portions of the central nervous system outside of the cord. Warburg, for

* Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 28, 1946.

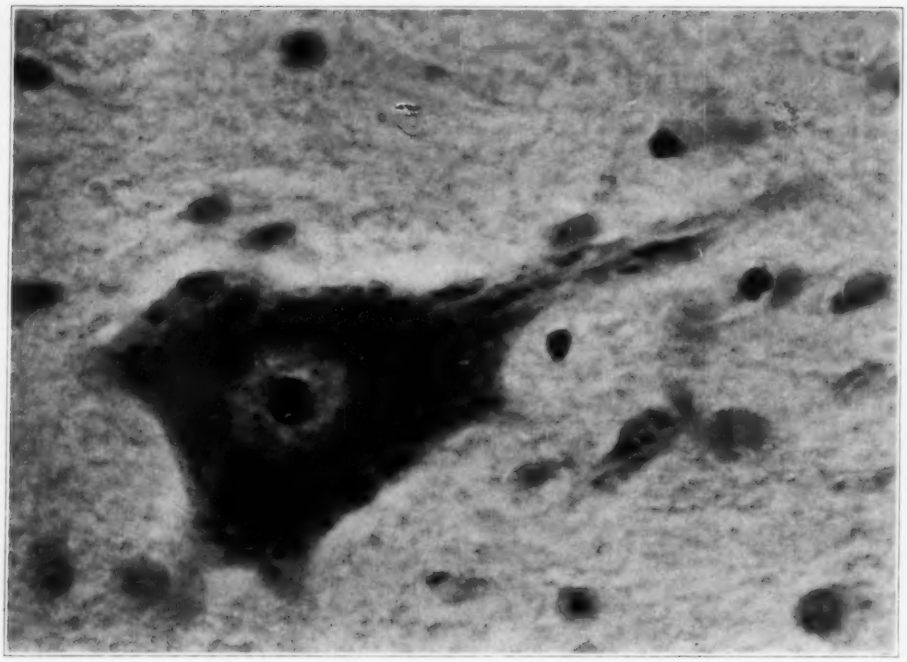

FIG. 1

Normal anterior-horn cell (× 792). Notice large Nissl bodies distributed throughout the cytoplasm, numerous cell processes with a few Nissl bodies in the axon, and the central location of the nucleolus.

instance, mentioned lesions in the cord and the spinal ganglia, the medulla oblongata, the pons, the mid-brain, and the basal ganglia, as well as the cortex and cerebellum, variously interpreted as inflammatory, degenerative, or regenerative. As far as the relative origin of these changes is concerned, Környey stated that, cranially from the medulla oblongata, the changes were mainly mesodermal and glial, which means interstitial, while the main seat of the parenchymal lesions remained in the anterior-horn cells. In the cortex, the mesodermal changes are the first sign of the poliomyelitic process, and there is a definite selectivity as far as the area and layer of the cortex are concerned,—namely, the large, or giant cell, fifth layer in the motor area[16]. In these higher regions, the infiltration of neuroglial cells and lymphocytes is far more extensive than the changes in the motor-nerve cells themselves.

Further evidence has been given of lesions cranially from the medulla to the mid-brain and the basal ganglia. The report of Peters on seventeen autopsies showed that changes occurred, not only in the anterior horn, but also in the posterior and lateral horns and in the intermediate zone (later called the internuncial pool) and cranially in the nucleus ruber, in the substantia nigra of the pons, and almost regularly in Deiters's nucleus.

The motor cells are more often and more extensively involved than the sensory cells, but this selective affinity of the virus for cells of the motor type is not absolute. Swan found the sensory ganglia—spinal as well as cerebral—the thalamus, and the hypothalamus involved. Sabin described the constant changes in the neural connections between the alimentary tract and the central nervous system.

Thus it becomes evident that, since Charcot's time, pathological studies have led to a gradual extension of our concept of the seat of the disease. This concept now covers many parts of the central nervous system, and it is much more adaptable for an explanation of clinical symptoms and course.

The selectiveness of the paralysis, and the prevalence of the partial over the total paralysis of muscles, to which Lovett has already called attention, suggest a spotty focal

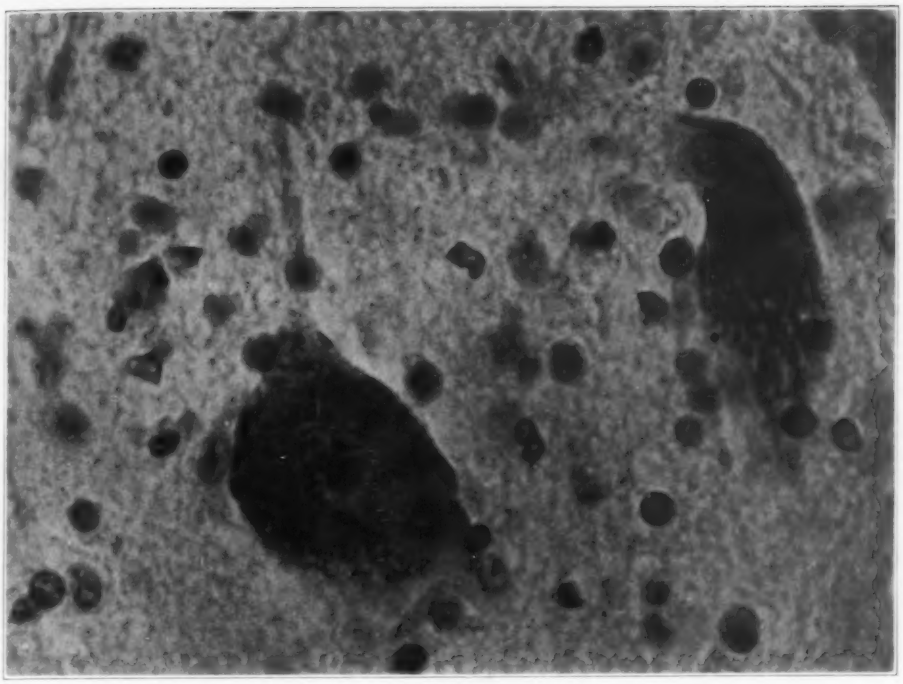

FIG. 2

Section of an anterior-horn cell (× 422), showing early central chromatolysis, in acute poliomyelitis. Nissl bodies are fragmented and have largely disappeared from the center of the cells. Microglia cells and lymphocytes are evident. Infiltration or inflammation in this region of the anterior horn is mild.

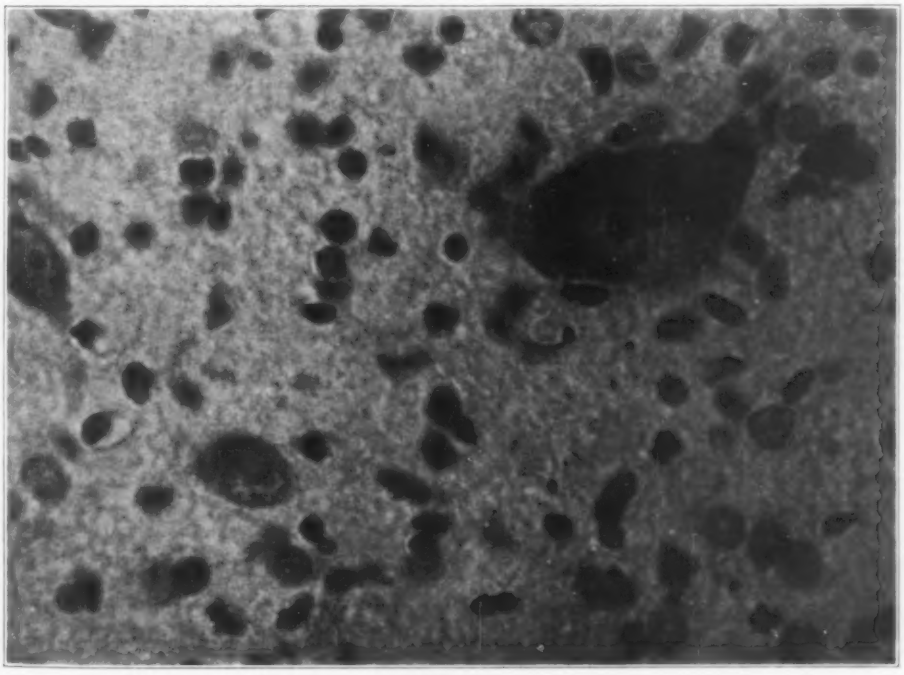

FIG. 3

Section (× 422) from the lumbar segment of the cord in a case of acute poliomyelitis, showing central chromatolysis of the anterior-horn cell, with fragmentation of the cell walls. Notice almost complete loss of the Nissl bodies and peripheral location of the nucleolus. This area of the anterior horn is densely infiltrated with inflammatory cells.

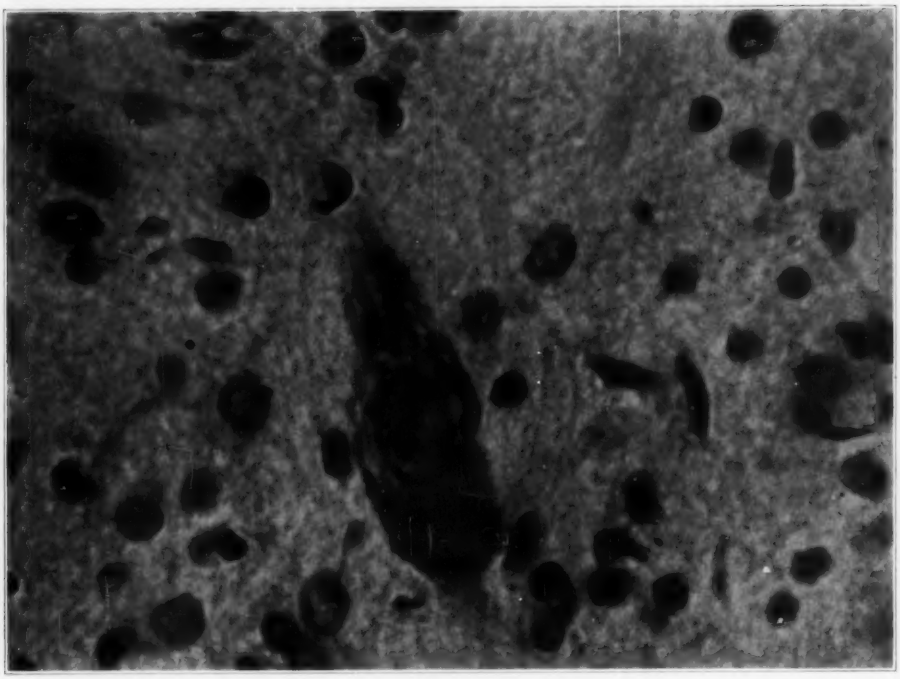

FIG. 4

Section (× 422) of an anterior-horn cell, showing early central chromatolysis, in a case of acute poliomyelitis. Same pathological changes are present as in Fig. 2.

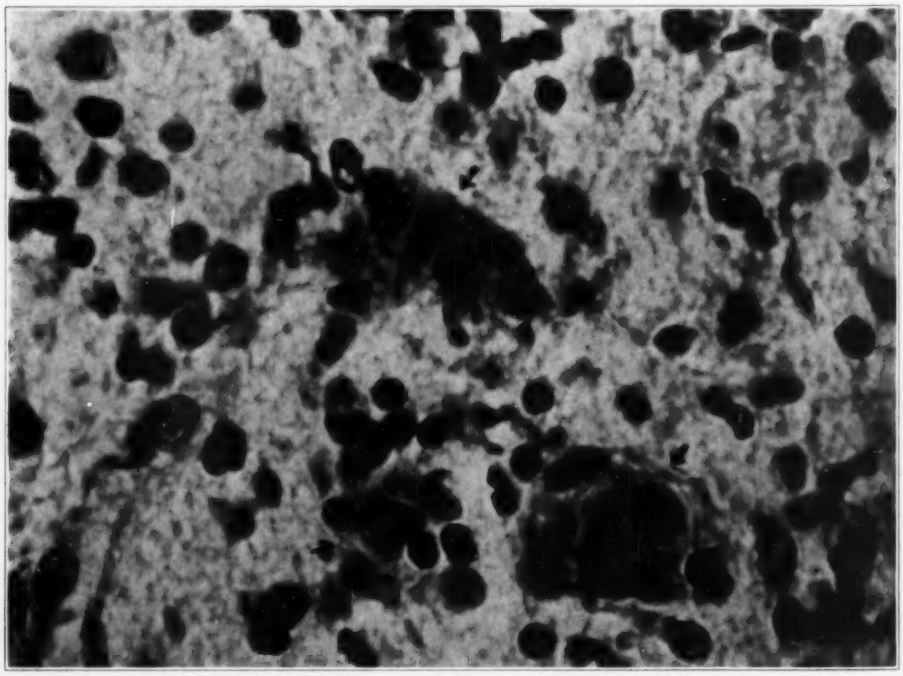

FIG. 5

Satellitosis of the anterior-horn cells. Three anterior-horn cells which have been fragmented are seen in this photomicrograph (× 422). The nucleoli and cell remnants are surrounded by monocytes or macrophages; microglia cells and lymphocytes are scattered throughout.

involvement of the cord. In cases of early arrested and non-paralytic poliomyelitis in monkeys, Bodian and Howe found minimum changes, which consisted of discrete regional

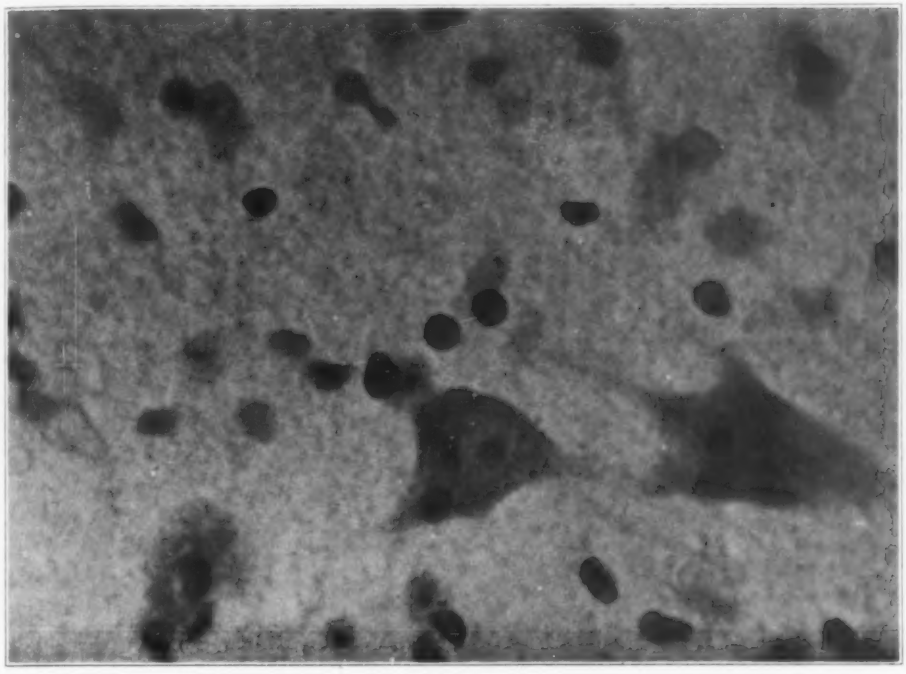

FIG. 6

Normal intercalated or internuncial neurons. These cells have fewer processes and smaller masses of chromatin matter, and are smaller than anterior-horn cells. (× 792.)

destruction of neurons with mesodermal glial infiltration and cuffing. The absence of clinical paralysis is due to the scattered distribution of the motor-neuron lesions.

Many years ago this so-called focal distribution[23] was studied from the anatomico-topographical point of view by Sano and Bruce.

In the cross section, certain clusters of motor cells appear in the cervical and lumbar intumescence in the lateral portion of the anterior horn, in addition to the constant and segmental clusters which occupy the more medial portions. As more muscle groups appear, these additional clusters are added in such a fashion that the more distal muscle groups have their nuclear representative in the increasingly lateral portions of the anterior horn; this is similar to the motor and sensory arrangement in the precentral and postcentral gyri of the cortex.

Of still greater interest is the perpendicular arrangement, in which these clusters appear as columns. It seems plausible that the destruction of a cell cluster in a given cross section may mean little or nothing to the function of the muscle, which is supplied by a certain nuclear column. The medullary nuclei of some of the muscles spread over a number of spinal segments, while others (for instance, the tibialis anterior) spread over a few only. This may explain why some muscles are more likely to escape complete paralysis than others.

Analysis of the 1000 cases of paralysis of the lower extremities, collected by Sher, shows that the tibialis anterior was severely involved in 83 per cent., the tibialis posterior in 79 per cent., the gastrocnemius in 62 per cent., and the peronei in 66 per cent., to mention only the muscles most frequently involved. On the grounds of the topographical extension of these columns, one may explain the relative frequency and degree of involvement of single muscles or of muscle groups. However, to the author's knowledge, Sano's diagrams have not yet been confirmed by serial sections of the cord.

The second point of clinical interest is recognition of the reversibility of the anterior-horn lesions (Fig. 1). Obviously, complete destruction of the anterior-horn cells is final

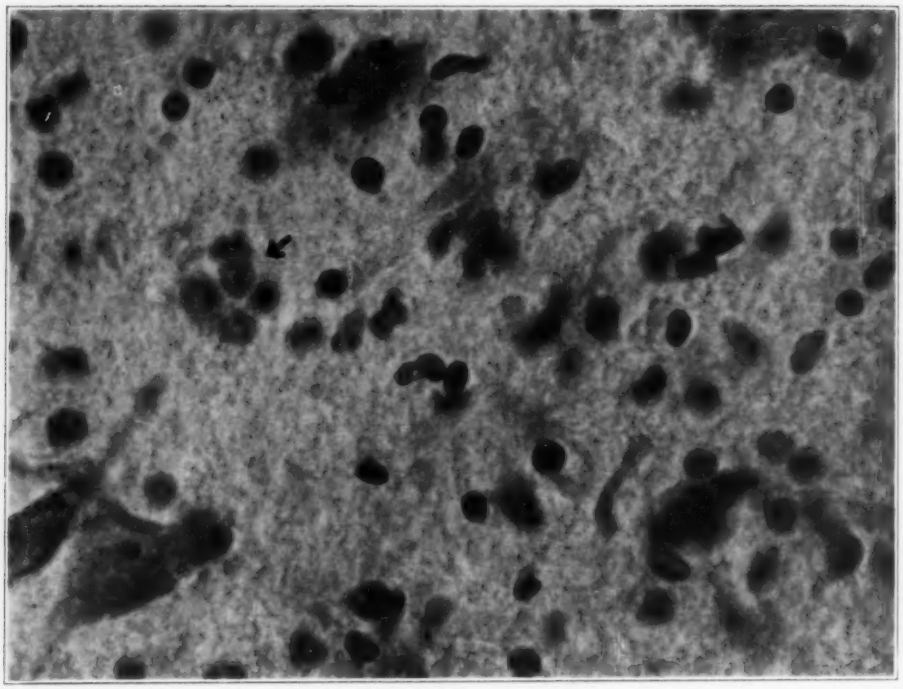

FIG. 7

Intercalated neurons, showing chromatolysis and satellitosis. Notice the marked chromatolysis of the neurons, with a moderate number of inflammatory cells present. The arrow indicates an intercalated neuron that has succumbed; only the nucleolus remains. Macrophages, like vultures, surround this remnant. (× 422.)

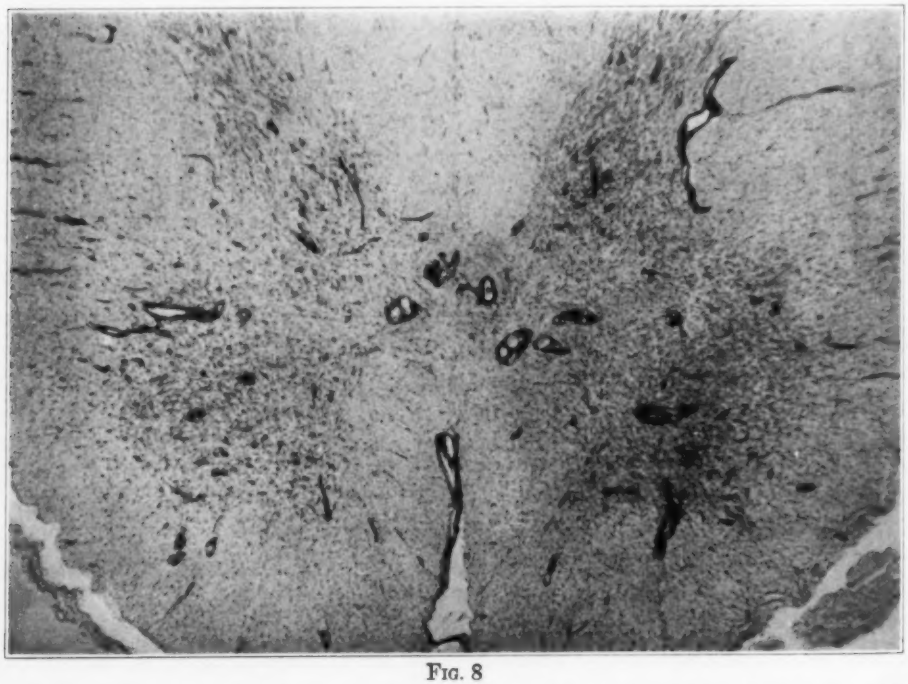

FIG. 8

Section from the lumbar cord in acute poliomyelitis. A low-power photomicrograph (× 106), showing the infiltration of microglia and lymphocytes and the perivascular cuffing which exist throughout the gray and white matter of the cord, in some areas. The pathological changes are not limited to the anterior gray column.

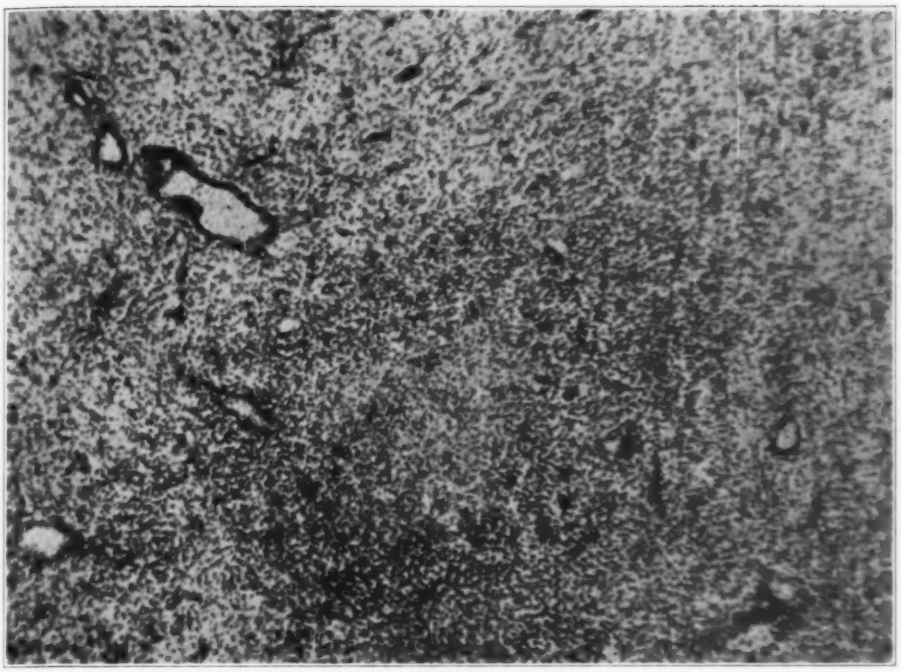

FIG. 9

Section (× 106) from the lumbar cord in acute poliomyelitis, showing necrosis and hemorrhage of the anterior horn. Notice the dense infiltration of inflammatory cells throughout this area. Tissue continuity has been lost in the central portion, due to necrosis, and hemorrhage has occurred.

and permanent damage; yet up to a certain point the changes seen in these cells must be reversible, just as the inflammatory glial infiltration and the perivascular lymphocytic exudates or cuffs are reversible. This is essentially the sequence of changes (Figs. 2, 3, 4, and 5) in the motor cells of the horn, which was known to Cadwalader in 1908 and Wickman in 1909: the chromatolysis, the sharply outlined acidophilic inclusion, or the Nissl bodies, and the peripheral arrangement of these bodies; then the loss of cell outlines, complete disintegration and crumbling of the cell, followed by satellitosis of the nucleus; phagocytosis; and finally gliosis.

Patton distinguishes three phases: The first is the early swelling of the cell with enlargement of the nucleus and disappearance of the Nissl bodies. The second is the chromatolysis with basophilic granules filling the cytoplasm. The final stage is the breaking up of the cells, with necrosis.

If there is a certain degree of reversibility of the changes in the motor cells of the horn as they exist in the secondary reaction of glial infiltration and perivascular cuffing, it furnishes a plausible explanation for the complete recession of paralysis, just as the focal distribution of poliomyelitis in the columns of motor cells in the anterior horn explains all degrees of partial paralysis.

The next clinical symptom to be explained on pathological grounds is the spasm, which has become the most controversial feature of all. It is now believed that spasm represents a dysfunction of the intermediate, or so-called internuncial, cell group of the anterior horn. Neurologists have known for a long time[21] that there is a synaptic relay group, both motor and sensory, situated dorsal to the cells of the anterior horn (Figs. 6 and 7). All impulses going through the spinal reflex arc, as well as those coming down from higher centers, are relayed through these cells to the motor neurons of the anterior horn.

Kabat and Knapp have attracted wide attention by a report on the pathological findings on not less than seventy-eight patients, who died as a result of poliomyelitis. They regularly found lesions of this intermediate cell group; and furthermore, in 40 per cent. of

FIG. 10

The lumbar cord following acute poliomyelitis, showing gliosis of the anterior horn. No semblance of the anterior horn remains. Scar of gliosis completely replaces most of the area. (× 100.)

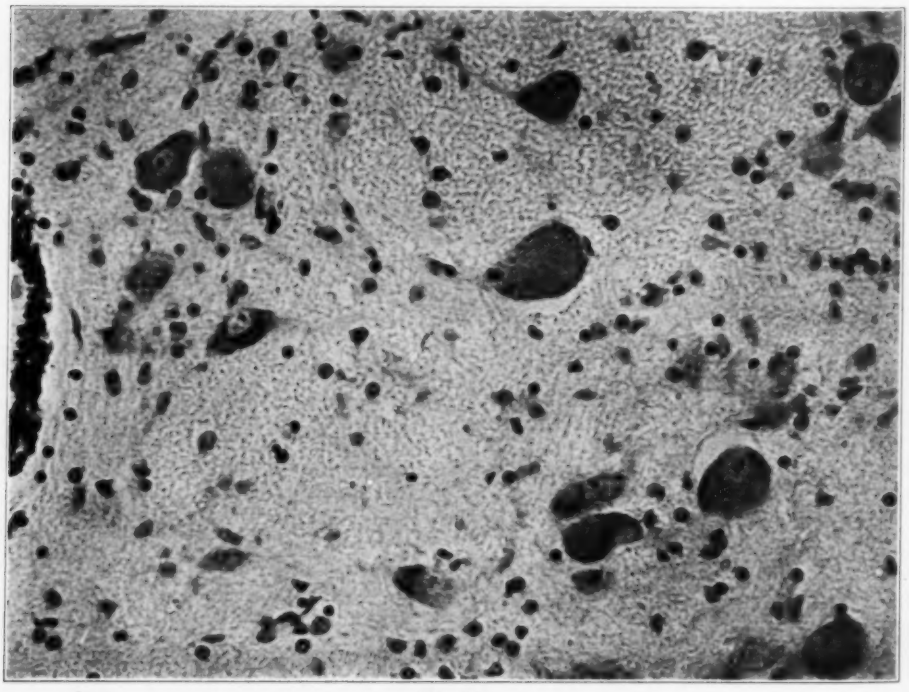

FIG. 11

Area of the medial vestibular nucleus in the floor of the fourth ventricle in acute poliomyelitis. Notice perivascular cuffing, central chromatolysis of the neurons, with peripheral location of the nucleoli, and infiltration of lymphocytes and microglia cells. (× 422.)

their cases, the internuncial cells alone were involved, while the cells of the anterior horn were intact.

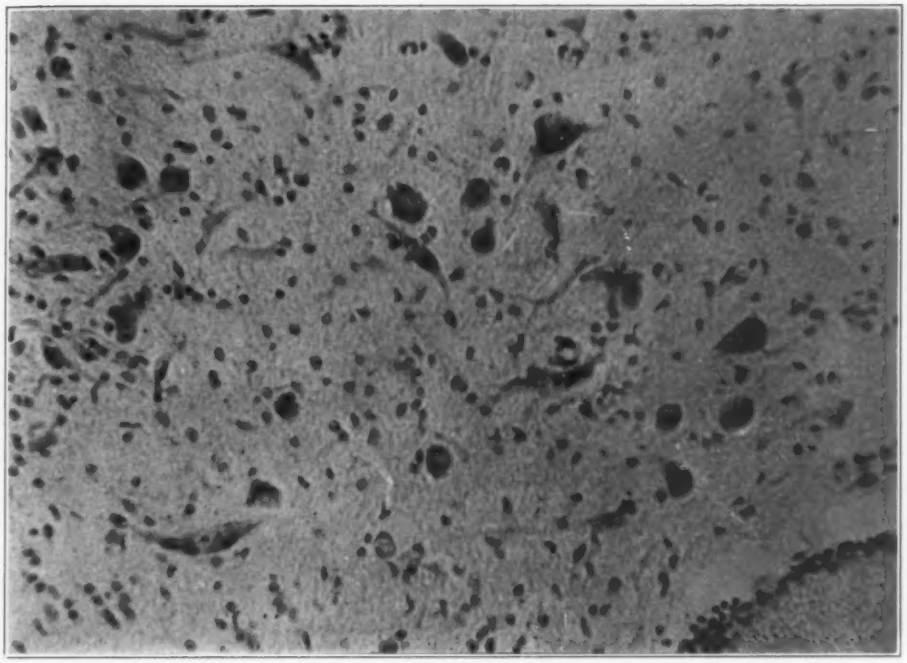

FIG. 12

Area of the medial vestibular nucleus in the floor of the fourth ventricle (× 422). The conditions are similar to those present in the case shown in Fig. 11.

Unless these findings are based upon serial sections, it would not necessarily mean that the anterior-horn cells have entirely escaped destruction, except at a certain level.

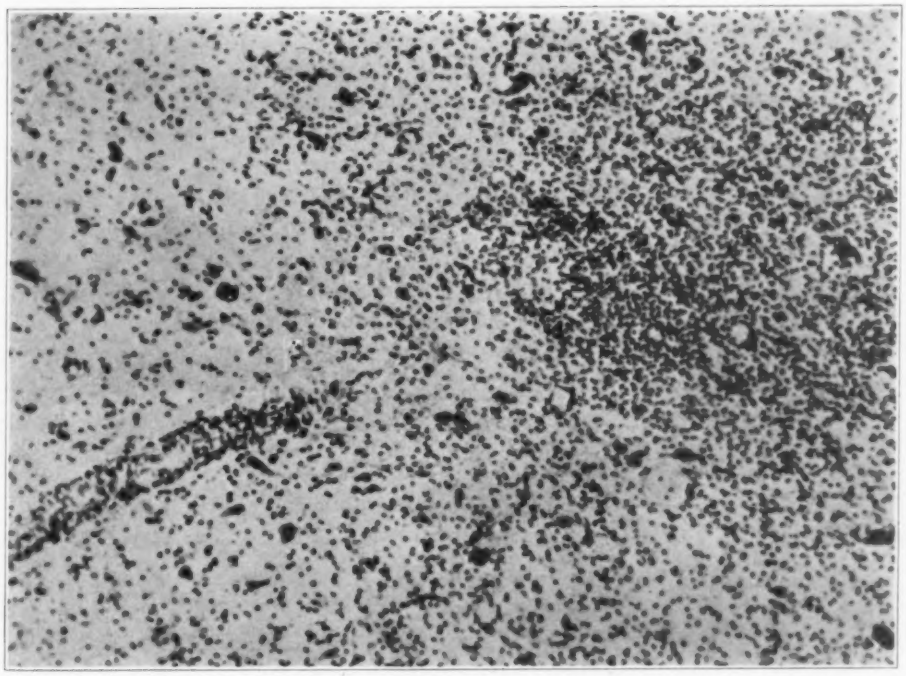

FIG. 13

Section (× 422) from the mid-brain in acute poliomyelitis. The same type of inflammation noted in Fig. 12 is present here, also. Notice the microglial and lymphocytic infiltration and the perivascular cuffing.

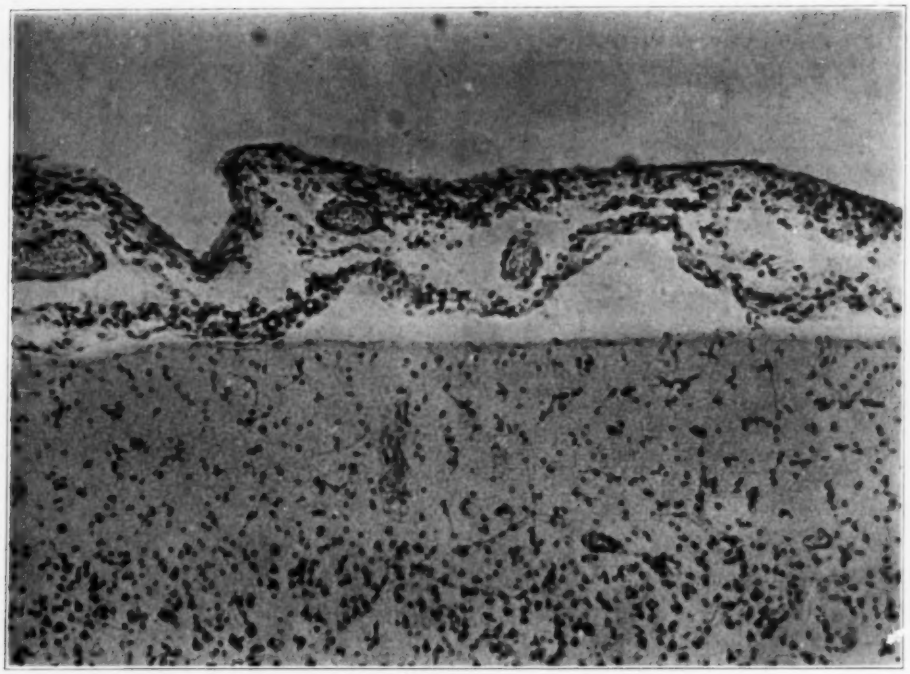

FIG. 14

Section (× 422) of the meninges of the brain in acute poliomyelitis. The meninges have become thickened by the inflammatory process. Dense infiltration of lymphocytes and mild perivascular cuffing are present.

In the author's small series, anterior-horn cells were found to be destroyed, either alone or together with the internuncial cell, but not the reverse (Figs. 8, 9, and 10).

The clinical corollary of this internuncial-cell lesion is believed to be the spasm. There is no doubt that it is a most frequent symptom, and that it is entirely different from contracture. During the 1944 epidemic in Iowa, the author found spasm in practically all cases, and almost invariably in the quadriceps. Even though the muscle was incapable of active contraction, it felt hard and indurated; and attempts to flex the knee caused pain, referred to the muscle and not to the joint. Thus, if we admit the role of the internuncial-cell group, spasm would appear not to be a phenomenon of the lower motor neuron at all, but would belong to the higher regulatory, or inhibitory, centers.

Consideration will now be given to the lesions in the higher centers,—namely, the medulla oblongata, the pons, the basal ganglia, and the more recently described changes in the tegmental regions of the mid-brain (Figs. 11, 12, and 13). Peers described, in the paracentral gyrus, perivascular cuffing, lymphoid infiltration, and thrombosis of the vessels. He observed that these changes, as well as the others in the tegmental nucleus, are reversible and transitory, and that they decrease in monkeys as time elapses. Bodian injected poliomyelitis virus intracranially into three monkeys, waited for spasm to develop, then killed the animals. He found lesions in the basal ganglia, but none in the cord. The occurrence of these lesions may well be in harmony with the phenomenon of incoordination and asynergic contraction of muscles—a disturbance of the normal reciprocal innervation—as is postulated by Sherrington.

It is not beyond the realm of possibility that the symptoms of so-called "mental alienation", as Miss Kenny calls it, represent the loss of voluntary cerebral control, a physiological block within the central nervous system, and, more specifically, in the system of synapsis located all the way from the cortex to the cord. While we recognize this as a transitory and reversible stage, it may for all practical purposes appear as a real paralysis. In contrast, however, to the changes due to the destruction of the anterior-horn cells, these

changes are entirely reversible; and this may well explain some of the early and remarkably rapid cures, now ascribed to our own handiwork (Fig. 14).

It must also be considered that not all cerebral lesions are necessarily reversible (Fig.

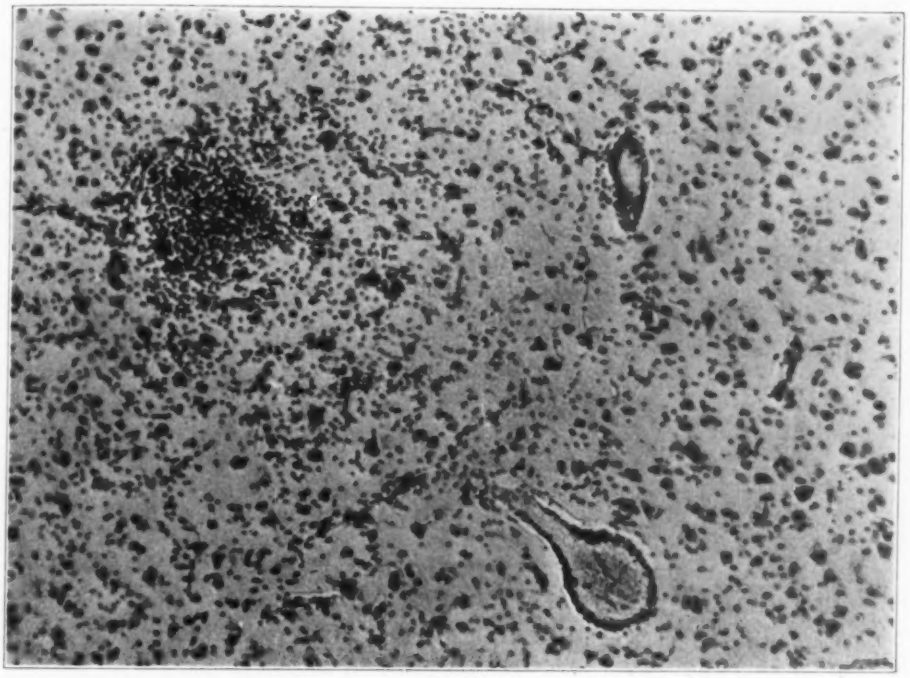

FIG. 15

Section (× 422) showing the fourth layer of the cerebral cortex in acute poliomyelitis. The same type of inflammation is present as in Fig. 14.

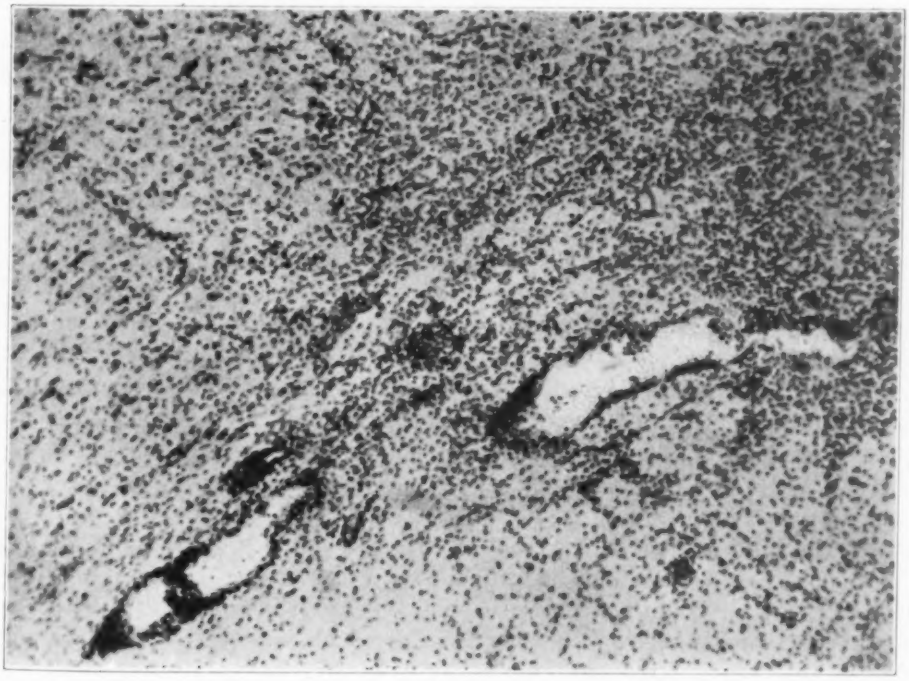

FIG. 16

Section (× 422) of the posterior horn of the cervical spine in acute poliomyelitis. Massive infiltration of microglia cells and lymphocytes is present, with perivascular cuffing.

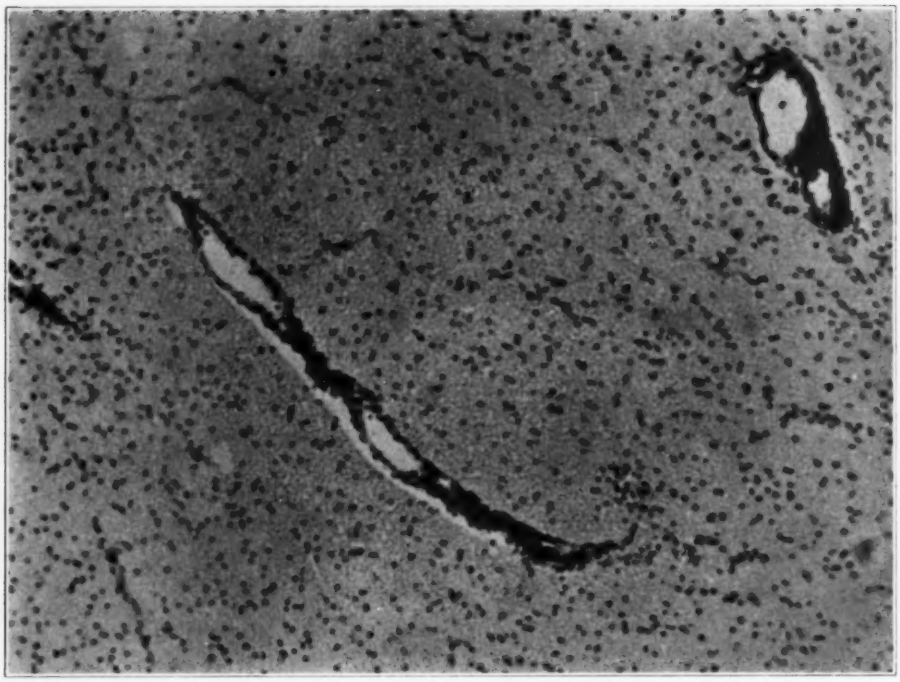

FIG. 17

Section (× 422) of the posterior horn of the lumbar area of the cord in acute poliomyelitis. Notice dense inflammatory reaction present in this portion of the cord.

15). This is illustrated in epidemics or solitary instances of infantile paralysis, where the residual symptoms are not paralysis or shrinkage of the muscles, but fatigue, incoordination, and lack of concentration,—features which point distinctly to the cortex and which make it difficult to draw a strict boundary line between poliomyelitis and polio-encephalitis.

The question arises of what is the pathological equivalent to the sensory symptoms of pain and skin irritability.

Small foci of infiltration of the dorsal root in the sympathetic ganglia have been recognized for decades. Sachs, on the basis of eight autopsies, described changes in the central gray matter,—evidently the internuncial cells, as well as changes of the posterior horn (Figs. 16 and 17). More recently, Toomey expressed the belief that the first cellular reaction is probably destruction of the cell elements of the spinal ganglia; and he also reported involvement of the lateral horn, which is subserving the autonomic system. From our clinical observations, these sensory lesions represent reversible changes. Whether or not true lesions of the sympathetic system exist is still undecided. From the clinical point of view, their occurrence is not unlikely. We refer here to the changes in circulation often seen in severe cases. From our own observations with sweating, and from other tests[41], hypohidrotic, vasodilatory, and hypopilomotor changes have been found. These are hard to explain, except on the basis of functional insufficiency of efferent sympathetic nerves.

PHYSIOPATHOLOGICAL STUDIES

Studies in physiopathology have been carried on in recent years, and the center of interest is held by the muscle and the neuromuscular junction.

However, John Ridlon, more than fifty years ago, pointed out that within a short time the affected muscle begins to become atrophied, and he stated that there is nothing in this change to distinguish it from the atrophy of disuse. This statement established the distinction between simple atrophy and degeneration, which is necessary in order to understand the subsequent relations of neuromuscular regeneration. That a muscle can recover

from an advanced stage of simple atrophy and that it could not recover from a similarly advanced stage of true degeneration has already been amply indicated. As Aycock aptly puts it, the doctrine of pleurisegmental innervation of the individual muscle fibers (in accordance with the topographical scheme of Sano) offers the most adequate explanation of the phenomenon of recovery.

The histological corrollary of regenerability of the muscle fibers has been studied. Simple atrophy shows a shrinkage of the fiber, and an apparent, but not a real, increase of the sarcolemma nuclei; later there is a disappearance, first, of the cross striation, and then of the longitudinal striation. It is believed that these stages are still reversible. The more advanced changes in the muscle begin with vacuolation, necrosis, and crumbling of the sarcoplasm, and proceed to proliferation of the interstitial tissue, and, finally, to fibrous or fatty substitution of the parenchyma. These latter changes are believed to be irreversible.

The next problem was to find the physiological responses which correspond to the different phases of muscle atrophy and degeneration.

Physiologists have been interested in the action current of the paretic muscles. Proebster made the significant finding that there was no parallelism between excitability and tension strength of the paretic muscles. The excitability may be 100 per cent. of normal, whereas the contractive strength may be only a fraction of it. Furthermore, Proebster spoke of the state of permanent excitation of the paretic muscle, which entails the danger of early fatigue. The poliomyelitic muscle may have as low a threshold of excitability—that is, response to small stimuli—as does the normal muscle, but the important point is that, when it does respond, it responds with a maximum oscillation.

It is of significance that, in true muscle degeneration, there is a retardation of the appearance of action current on voluntary contraction; but this could not be demonstrated by Proebster in the majority of poliomyelitic muscles, which evidently were in the state of simple atrophy. The paretic muscle of poliomyelitis, on continuing voluntary innervation, produces action-current curves which correspond to maximum effort, and consequently are likely to lead to early fatigue. In this latter case there appears, then, the picture of incoordinated action-current curves, with low amplitudes and exhaustion.

These earlier action-current experiments are mentioned because they fit in so well with the recent investigations carried out by Bouman and Schwartz. These authors showed in the electromyograms that spasm exists not only in the antagonist, but also in the weakened muscles. However, spasm does not exist in the totally flaccid paralyzed muscle. Furthermore, Bouman and Schwartz found that the paretic muscle-current potential increases with the improvement in motor power. These findings seem to prove that spasm is a general phenomenon in infantile paralysis, occurring in the earlier stages of the disease. It appears in the synergist and antagonist alike, and has nothing to do with denervation of the motor horn. It finds a plausible explanation in the dysfunction of the internuncial-cell group, which has lost its inhibitory effect on the spinal reflex mechanism.

The understanding of the neuromuscular dysfunction of the poliomyelitic muscle has been further advanced by experiments on chronaxia, which means the time necessary for a current of standard intensity—that is, twice the minimum rheobasic current—to produce muscle responses. These experiments are not new. As early as 1928, Marinesco, Sager, and Kreindler conducted such investigations on muscles in infantile paralysis. Péhu and Morin found in eight cases, during the first days of poliomyelitis, a moderate increase of chronaxia values in the affected muscles, which values reached their maximum in a few weeks. Then, as the muscle recovered, the values came down in from two to eight months. The time of evolution and the intensity of these chronaximetric disturbances were found to be proportional to the severity of paralysis. Also, Chauchard and his associates found that, in monkeys, the chronaximetric changes appeared earlier than the paralysis, which indicates an early encephalitic involvement.

Foremost in the investigations along this line stand those of Moldaver, who emphasized the electroprognostic value of chronaxial measurement in this disease; he believed that these measurements offered the best index of functional recuperation. He showed that muscles in spasm have normal chronaxia values. Neuromuscular degeneration can be determined by chronaxia, while spasm does not lead to neuromuscular degeneration. The muscles with only slightly increased chronaxia time have a good prospect of recovery, while those with prolonged chronaxia or those not responding at all offer a poor prognosis.

Recently, experimental physiology has taken another helpful step in the interpretation of symptoms concerning muscle tone and muscle volume. We have known that fatigue is under the influence of the central nervous system and it is not connected with the neuromuscular unit itself,—at least, in the healthy muscle. Proebster has already pointed out that the considerably atrophic and shrunken poliomyelitic muscle requires, even for a moderate contracting effect, a maximum expenditure of energy. Therefore, fatigue will set in much sooner in such a muscle.

The impulses, which in the normal muscle are imparted sufficiently by the cord or by higher reflexes, require in the paretic muscle a sustained and conscious energy effort; hence early fatigability results. In addition, the experiments of Orbeli showed that fatigue can be retarded by stimulation of the sympathetic nerves.

Orbeli's experiments do not, however, parallel the clinical situation, which can be correlated much better with more recent experimental findings. These findings have changed former ideas about the fatigability of weakened muscles, and it is no longer believed that absolute immobilization is necessary to save a weakened muscle from exhaustion.

The experiments of Hines and his associates at Iowa University [15,22,51] showed that, following denervation, the failure of the muscle to develop effective tension is the cause of atrophy; they also found that inactivity and immobilization retard neuromuscular regeneration, while, on the other hand, a program of early muscle use promotes it. These authors further found that electric stimulation, of the order required for maximum tension and for maximum physiological stretch, greatly retarded the loss of weight and the loss of strength of the muscle in the period which preceded its re-innervation. Finally, they found that not even the resultant fatigue seemed to have an injurious effect on any phase of neuromuscular regeneration.

The application of these newer discoveries to the clinical interpretation of symptoms and to the policies of treatment is far-reaching.

Because of the high incidence of spontaneous recovery, any therapeutic measure would be hard to evaluate. In addition, improvement in muscle function may not all be due to recovery of a nerve cell from shock or other reversible change within the cell itself, but it may be due to hypertrophy of the remaining uninvolved muscle fibers.

From the standpoint of treatment, spasm is considered to result from loss of the inhibitory action of the internuncial-cell group and of the higher centers. With this idea in mind, Kabat and Knapp introduced prostigmine, because they believed that it not only reduced the hypertonus of the skeletal muscles and the proprioceptive reflex hyperirritability, but it also reduced the incoordination, which would indicate that this drug acts upon the higher centers, also.

Prostigmine may not find general use in poliomyelitis, since the spasm and the hyperirritability are transitory symptoms. The author's experience is too limited as yet to justify on opinion; but, from the experimental side, Hines and his associates found neither a delay nor a hastening of the neuromuscular regeneration after prostigmine injection.

Physiological Effect on Nerve Regeneration

A recent plan to promote nerve regeneration is the so-called nerve crushing, or neurotripsy. The idea is not entirely new. In 1935, Iovino produced, experimentally, paralysis of the sciatic nerve by cutting the nerve partially and then resuturing the cut end. Within

three months the function of the leg reappeared; it became normal in six months, and the muscle atrophy disappeared. Iovino reported that after ten months there was an actual increase in the sutured nerve fibers over those of the normal sciatic nerve on the other side. The regenerated surplus nerve fibers were uniformly distributed in the entire peripheral end of the sectioned sciatic nerve.

In line with this concept of hyponeurotization, Billig, Van Harreveld, and Meersma studied the regeneration of nerves after nerve crushing. Experiments were carried out on rabbits. The quadriceps was made equally paretic on both sides by pulling out some of the spinal roots of its nerve supply. The authors then proceeded to crush the remaining fifth lumbar root on one side, leaving the root on the other side intact. They found that, after the crushed root had regenerated, there was actually an increase of muscle power on the side of crushing over that on the other side. The underlying idea was that the regeneration after crushing not only equals, but actually exceeds, the supply of axis cylinders and neuromotor end-plates over and above the supply present before the crushing.

This principle was then applied to patients with partially paralyzed muscles; the crushing of their motor nerves was done either openly, near the motor points of the muscle, or subcutaneously, with an instrument which resembled a compressed-air riveter. The improvement of the muscle power was carefully measured, according to a standard grade scale and by electromyograms. A number of patients who have been interviewed by the author expressed themselves as having definitely been benefited by the procedure. According to Hines and his associates, the maximum recovery after crushing, measured by muscle weight and tension, is about 85 to 90 per cent. It is remarkably constant, and reaches its peak in about twelve weeks. Nevertheless, no admission has yet been secured from physiologists that, following nerve crushing, the amount of regenerated nerve supply exceeds or even equals the normal.

TREATMENT

In summary, these more recent studies in the pathology and physiopathology of muscle and peripheral nerves do not seem to have brought about anything resembling a complete and radical change in the management of anterior poliomyelitis; they have, however, produced important modifications in our concepts of conservative treatment.

Rest

The application of heat and rest and the avoidance of stretch reflexes in the affected muscles are still being adhered to, along the lines laid down by Lovett; in addition, during the period of hyperaesthesia, all limbs are kept in proper position. The technique of heat application has been vastly improved by Miss Kenny, and the results are particularly striking in such conditions as intercostal rigidity, spasm of the neck and back muscles and of the hamstrings, and quadriceps rigidity.

Immobilization

Casts are almost never used, and braces for the protection of joints are applied only to prevent deformity. Weight-bearing is allowed as early as possible, and recumbency is prolonged only in patients threatened with spinal asymmetry.

Movement

It is especially important to keep up the muscle tone by early massage and by systematic active and passive movement. The fear of exhausting or permanently damaging a muscle by taking it through the full range of motion is, in the light of experimental evidence, without foundation.

It has been established that early mobilization favors, rather than impedes, neuromuscular regeneration. This applies to the so-called "alienated muscle", which Moldaver has shown, on grounds of chronaxial changes, to be the one really affected. There are,

however, more central inhibitions of voluntary motions which prevent active contractions temporarily, and these muscles offer the best prospects for re-educational methods.

The same may be said of the mass movement called incoordination. The muscle must be taught to resume its proper function,—first by passive motion, which brings out the proprioceptive stimuli, and then by active motion, which re-establishes and fixes the motor pattern in the brain. It is clear that only a weakened, and not a completely paralyzed, muscle can be expected to respond to such educational treatment.

DISCUSSION

The impression should not be gained that there is any kind of supremacy of the laboratory worker over the clinician. There are too many discrepancies between the situation existing in animal experiments and that obtaining at the bedside to make any laboratory results absolutely binding; on the other hand, the clinical observations are so numerous and have been made by so many competent and experienced physicians that they cannot be rejected simply because they do not harmonize with experimental findings. We cannot, however, be blind to the fact that, in many perplexing problems concerning anterior poliomyelitis, experimental medicine and basic medicine have opened our eyes to certain traditional mistakes and misconceptions which will have to be corrected or revised.

NOTE: The writer wishes to acknowledge, with thanks, the valuable help of Dr. W. R. Ingram, Professor of Anatomy, State University of Iowa, in the selection and reproduction of the pathological slides.

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DISCUSSION

Dr. H. Relton McCarroll, St. Louis, Missouri: Dr. Steindler has given us a complete and excellent review of the experimental and clinical material which has contributed to our present knowledge of the pathological lesions associated with poliomyelitis. These lesions are widespread, as he has stressed, and are found during the acute stage of the disease. They are not to be construed as playing any significant part in the final picture which these patients present. The great majority of these lesions are transitory in character. While they undoubtedly play a part in explaining the acute illnesses of these patients and the incidence of early mortality, it has yet to be shown that any lesion except the destruction of the motor cells of the anterior columns plays any part whatever in the extent of the permanent paralysis. A residual encephalitic type of involvement is occasionally encountered with poliomyelitis, but this merely represents a more widespread distribution of the virus in the central nervous system. As clinicians, our primary concern still remains the amount of residual paralysis which our patients show.

Dr. Steindler's conclusions with regard to treatment are to me the most interesting part of his paper. Lack of immobilization, institution of early weight-bearing, the use of early active and passive motion—or, in other words, early mobilization of the extremity and of the patient—are all suggested. This is essentially the same type of therapy received by that group of patients who have come to us several months after their acute illnesses, and who had never seen a doctor or a physical therapist. These patients had received no treatment whatever, had been allowed early free activity, and had been put on their feet as early as possible by mothers who feared that to keep them in bed would perpetuate the paralysis. Those of you who question that management of this type can also give very gratifying results—results with as high a percentage of recovery as accompanies intensive therapy—might alter your opinion if you gave the method a trial. The cases followed by Sherman and Hatcher at the University of Chicago have added further proof to this point.

Can we attribute any part of the success or failure of recovery in our patients to the treatment which is given? I do not think so. On the contrary, these results are dependent on the virulence of the infection, the resistance of the individual, and, in brief, the extent of the initial destructive lesion. We shall rightfully continue the treatment of acute poliomyelitis, but we must admit that it does not alter the extent of the initial destructive lesion or the residual involvement which our patients show. I have the greatest respect for the author of this paper and for his opinion, but it seems strange to me that we should still interest ourselves in attempting to rationalize a treatment which is doomed to failure before it is ever instituted.

Most of this paper has dealt with experimental work, and this prompts me to repeat a statement which I have made previously. After the destruction of anterior-horn cells has occurred, no form of therapy can alter the ultimate residual picture which these patients present. To accomplish this, the control must be brought about by prevention of the initial infection or by abortion of the process before cell destruction takes place. The one hope which we have at present of ever realizing this goal lies in the fields of epidemiology and of immunology. Today, only a small percentage of the vast sums spent for poliomyelitis work in this country is spent for investigative work in these fields. If we can see to it that funds and efforts are directed into these channels, we will then, and not until then, be able to entertain some hope of ultimately arriving at a solution to our problem. Our continuing to place so much emphasis on the treatment of poliomyelitis detracts from interest in other fields of investigation, which offer greater hope of real accomplishment.

Dr. William T. Green, Boston, Massachusetts: We would like to express our appreciation to Dr. Steindler for a scholarly discussion of the neurophysiological and neuropathological background of the treatment of anterior poliomyelitis. It is well to emphasize, however, that many of the concepts in this field are hypothetical and not subject to direct proof at this time.

Although the histological effects of the disease are widespread in the nervous system, I should like to reiterate that the involvement of the anterior-horn cells by the virus is the outstandingly significant pathological process. The cells may be affected in varying degrees—from complete destruction to minimum histological changes—and the evidence is that the functional effects may vary from a temporary cessation of axonal conduction to permanent and unalterable paralysis,—that is, up to a certain point the alteration of the anterior-horn cells by the disease is reversible.

Less important manifestations are not all explained by the direct effect of the disease upon the anterior-horn cells, although the expression of most of these abnormalities is in the distal reflex arc.

Muscle sensitivity and spasm and the disorder in reciprocal innervation are in this category. Regarding these manifestations, we should recall the diffuseness of the pathological changes in the nervous system, including the posterior ganglia, and the multiple synaptic connections affecting the anterior cells, both proprioceptive and cortical, either directly, or by way of the internuncial neurons. Disturbance of one or more of these pathways seems the likely explanation of the muscle spasm.

However, these features of the disease are not the important consideration. Spasm alone causes no significant residual disability, although, combined with paralysis, it contributes to deformity; and in so far as deformity occurs, it may affect the function of those muscles which oppose deformity. Sensitivity and spasm, however, do inhibit muscle function to a certain extent during the acute phase, but this is not peculiar to infantile paralysis.

"Mental alienation" is a term I prefer to avoid. Muscles to which such a term has been applied have been found to show varying degrees of neuromuscular degeneration.

I shall not discuss therapy except to say that (1) after the early phase, casts and splints are of great assistance in a certain percentage of patients; (2) I agree that complete immobilization impedes recovery, but so does overactivity in those significantly involved; (3) braces are not a scourge to be avoided, but should be used as indicated.

Dr. Arthur Steindler, Iowa City, Iowa (closing): In answer to Dr. Green, it is not enough to repeat again and again the story of the anterior-horn cells. The question is: Is there enough evidence to recognize reversibility of symptoms to the extent of spontaneous clinical improvement? It is obvious that, if improvement can occur spontaneously, there is no reason for giving credit to any particular kind of modality.

I did not state in the paper that I never use casts. I said that casts are practically never used. I use them very rarely. If we compare what we have done with what everybody else has done, we will find the results are about the same. I am very grateful to Dr. Green for his discussion. I think he has agreed with me.

I am sorry I cannot show evidence that action currents have relation to changes in chronaxia. Investigations show conclusively, however, that muscles which seem to be paralyzed are still active, and that completely paralyzed muscles never show action currents. As far as we know, no current is derived from a muscle in absolute rest.

In regard to Dr. McCarroll's discussion, I have a paragraph in my paper where I mentioned the report by Dr. McCarroll and Dr. Crego. There is no question that a lot of treatment is still being given undue credit which rightly belongs to the Lord, and not to any orthopaedic surgeon or physical therapist. I would not go so far, however, as to say that any sort of treatment of the muscles is useless.

THE DIAGNOSIS OF MENISCUS INJURIES

Some New Clinical Methods *

BY A. GRAHAM APLEY, F.R.C.S., PORTSMOUTH, ENGLAND

INTRODUCTION

In the year 1803, William Hey of Leeds wrote of a condition which he termed "internal derangement of the knee". This was not a clear-cut entity, but it did include the first, somewhat vague, description of locking; and Hey suggested that locking might be due to a meniscus lesion. However, as he treated his patients by manipulation alone, direct confirmation was not obtainable. The literature records hardly any progress in diagnosis for almost one hundred years, but the succeeding twenty or so years contain a galaxy of great names.

First Sir Robert Jones and later D'Arcy Power, Martin, and Morison recorded the results of numerous meniscectomies, and focused attention on the longitudinal split as the primary lesion. Then, in 1924, Bristow[1] opened a discussion on internal derangement of the knee at a meeting of The British Orthopaedic Association; and since that time Bristow[2], Platt[8], McMurray, and others have described large series of cases of knee injury, with careful analyses of their findings. Despite the meticulous work in all these series, one feature stands out,—namely, the high proportion of cases in which *no* meniscus split was seen at operation. Sir Robert Jones quotes a figure of 10 per cent., while Bristow found that in 30 per cent. of his cases there was no split, this proportion including many normal and hypermobile menisci.

Many attempts at an explanation have been made, but as recently as 1930 Platt was driven to conclude that this group remained an enigma. However, by this time the rotation sprain of the knee, in which the attachments of the medial meniscus to the tibia, capsule, or tibial collateral ligament were damaged, had also been described; and various authors had already emphasized the difficulty in distinguishing a rotation sprain from a split meniscus.

With all these advances, two notable gaps remain: first, the absence of any constant and reliable pathognomonic sign for a split meniscus; and, second, the difficulty of differentiating a split meniscus from a rotation sprain. The purpose of this paper is to describe some methods which aim at filling these gaps.

THE CAUSAL FORCE IN MENISCUS DAMAGE

The causal force in meniscus damage is clearly recognized. With the knee flexed and bearing weight, a twist occurs. Since the meniscus is fixed between the tibial and femoral condyles, if excessive rotation takes place, something must give way. Sometimes this grinding force splits the substance of the meniscus, and from this primary tear secondary extensions may occur, producing the different varieties of torn meniscus. Precisely the same force may, instead, wrench the meniscus away from some of its peripheral attachments, thus producing the so-called rotation sprain. It is quite true that weight-bearing (and therefore a grinding force) is essential in the production of a split in the meniscus, whereas it need not be present for a rotation sprain to occur. The important point, however, is that the selfsame force may, on occasion, produce either type of injury; and therefore the history may not help in differentiation.

* Read at the Spring Meeting of The British Orthopaedic Association, Newcastle-upon-Tyne, May 1946.

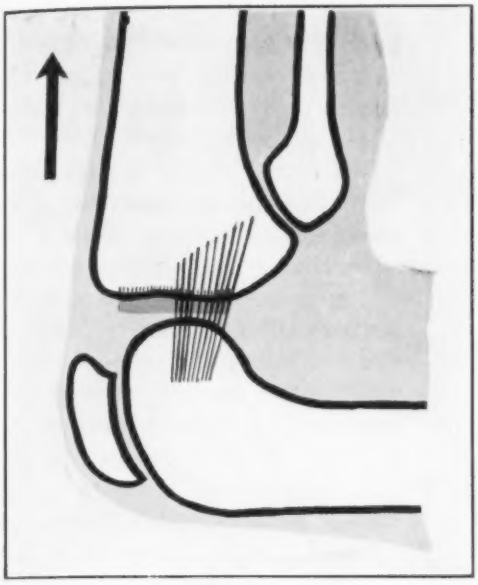

FIG. 1
Distraction of the knee.

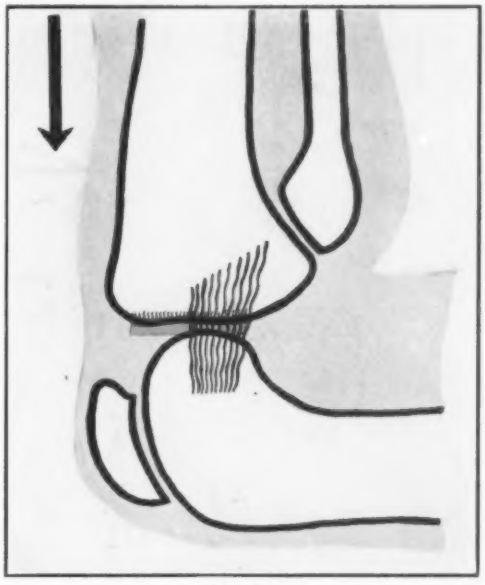

FIG. 2
Compression of the knee.

THE PRESENT METHODS OF EXAMINATION FOR MENISCUS INJURY

The most important feature in the present-day method of examination for a meniscus injury is rotation of the tibia, while the knee is held in varying degrees of flexion, as, for example, in McMurray's test. This manoeuvre is calculated to displace a meniscal fragment and to produce the well-known click, which is palpable, constant in position, and recognized by the patient. Together with the history (which, although of the highest importance, is not dealt with in this paper), a diagnosis can usually be made. All too often, however, signs are absent, and there seem to be three disadvantages in this method of examination:

1. A click is not a truly reliable sign. Its cause may lie in a different knee injury or in a different joint entirely. Many surgeons fail to find it at all, although McMurray declares that the diagnostic click is reliably constant in his personal cases.

2. The routine method of testing by rotation must necessarily pull on those tissues damaged in a rotation sprain, so producing extraneous pain. Thus, in the very process of testing for a meniscus injury, the surgeon may actively confuse the picture.

3. The third disadvantage, as Platt[9] has emphasized, is that the present method is based upon unstable mechanics. With the patient lying on his back and the surgeon examining his knee, there are two fixed points,—the pelvis, which rests on the bed, and the ankle, held in the surgeon's hand. Between these two, the long connecting levers of the thigh and the leg are unstable, and are not adequately steadied by the surgeon's hand.

A THEORETICAL SOLUTION

From the foregoing paragraphs it is evident that, in the examination of the knee, we are dealing with a confused problem of mechanics. To solve this problem, it must be resolved into its distinct components. First, stabilization of the levers can be accomplished by laying the patient on his face and fixing his femur in a way which will be described later. Second, to effect a diagnostic separation between meniscus and soft tissues, the application of a *longitudinal* stress is required. This stress should, alternately, be a separating (or distracting) force, and a compressing force; and these two types of longitudinal stress should be capable of easy application.

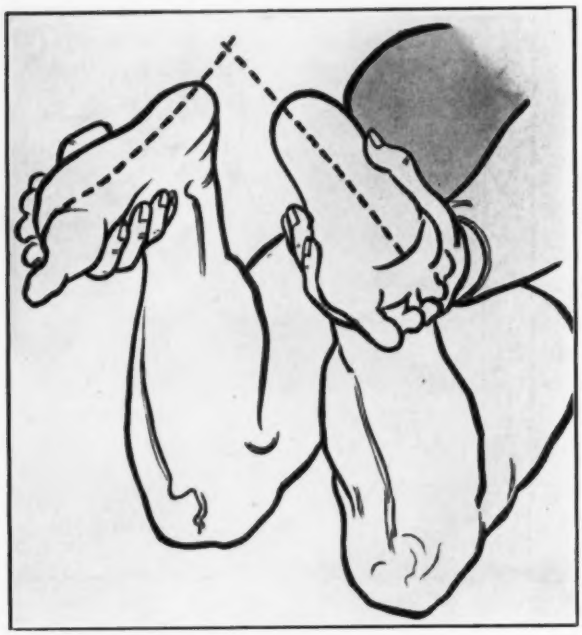

FIG. 3
Rotation of both knees—preliminary manoeuvre.

(The various illustrations epitomize both the theoretical arguments and the practical tests. These tests are easy to demonstrate in the patient, but less so in print.)

Figure 1 shows the theoretical result of applying separation or *distraction* to the knee. Clearly, the soft tissues are being stretched, while the meniscus remains undisturbed. (For simplicity, only the tibial collateral ligament is shown in the diagram, but the coronary fibers and the capsule must undergo similar stretching.) A test based on this fact is, therefore, a test of the soft tissues (ligamentous, fibrous, and capsular) and of these alone. Such a test would be positive in a rotation sprain.

Figure 2 illustrates the basis for a *compression* test. Here the soft tissues are clearly relaxed, but the meniscus itself is being crushed. In this position, in addition to the compression already produced, rotation can be added. This combination of compression and rotation constitutes a grinding force which is an accurate reproduction of the original destructive force

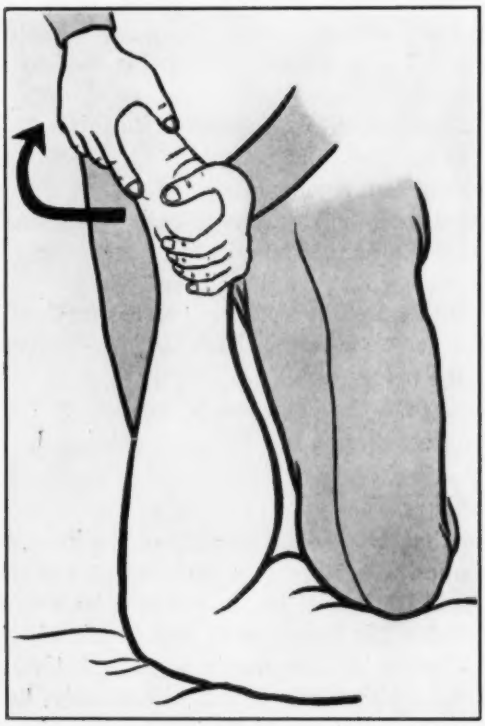

FIG. 4
Rotation alone.

FIG. 5
The distraction test.

in meniscus damage, so that a positive *grinding* test points unequivocally to meniscus injury.

Expressed in these simple terms, the two manoeuvres of compression and distraction form the basis of the proposed tests to fulfill the requirements previously put forward. It remains to translate these theoretical considerations into practice; and, to do this, a posterior method of examination is employed.

THE POSTERIOR EXAMINATION OF THE KNEE

For this examination the patient lies on his face. He should be on a couch not more than two feet high, or the tests become difficult, and he must be well over to the edge of the couch nearest the surgeon. To start the examination, the surgeon grasps one foot in each hand, externally rotates as far as possible, and then flexes both knees together to their limit (Fig. 3). When this limit has been reached, he changes his grasp, rotates the feet inward, and extends the knees together again. This preliminary manoeuvre demonstrates limited rotation, painful rotation, and the exact angles of flexion at which these occur; the estimation of these angles proves useful later in the examination.

The surgeon then applies his left knee to the back of the patient's thigh (Fig. 4). It is important to observe that in this position his weight fixes one of the levers absolutely. The foot is grasped in both hands, the knee is bent to a right angle, and powerful external rotation is applied. This test determines whether simple rotation produces pain.

FIG. 6
The grinding test.

Next, without changing the position of the hands, the patient's leg is strongly pulled upward, while the surgeon's weight prevents the femur from rising off the couch (Fig. 5). In this position of distraction, the powerful external rotation is repeated. Two things can be determined: (1) whether or not the manoeuvre produces pain and (2), still more important, whether the pain is greater than in rotation alone *without* the distraction. If the pain *is* greater, the distraction test is positive, and a rotation sprain may be diagnosed.

Then the surgeon leans well over the patient and, with his whole body weight, compresses the tibia downward onto the couch (Fig. 6). Again he rotates powerfully, and again he asks two questions: (1) "Does it hurt?" (2) "How much does it hurt?" If the addition of compression has produced an increase of pain, this grinding test is positive, and meniscal damage is diagnosed.

Incidentally, this question of the amount of pain is not a matter of fine hairline distinction; the patient must be sure of a considerable difference, and indeed he usually is.

THE USES OF THESE TESTS

1. So far, attention has been focused on differentiating a meniscus injury from a rotation sprain. This is the first, and perhaps the most important, use of these tests. They

are also useful in other types of cases, however, in some of which it is necessary to introduce modifications.

2. In many patients the history lacks diagnostic precision and the common physical signs of meniscus damage cannot be elicited; the findings are vague, although the patients may be grossly disabled. Again, in many patients the diagnosis of a lesion of the posterior horn of the meniscus may be difficult. In both types of cases, surgeons are not unreasonably chary of operating in the absence of any single clear diagnostic feature in the history or the physical examination. Not infrequently, therefore, the patient limps his way, for months or years, with the aid of physiotherapy. There is, therefore, a second use for the tests; for, if the grinding test is positive, then the doubt as to the diagnosis (and incidentally the meniscus) should be removed.

With a suspected tear of the posterior horn, the grinding test is modified; instead of holding the leg at a right angle, the knee is flexed much more acutely. The importance is now apparent of determining, in the preliminary manoeuvre (Fig. 3), the angle of flexion at which pain is produced by rotation, for the test should always be repeated at precisely this angle. In this way it may be possible to obtain accurate localization of the meniscus lesion as well.

3. A third use of the tests is in the diagnosis of lesions of the lateral meniscus, which may also be difficult at times. For this purpose the tests are modified by rotating the foot inward instead of outward; in other words, a *reverse grinding* test is performed. The clue as to whether this is worth trying is again given by the preliminary manoeuvre, in which *both* rotations at all angles of flexion were performed.

LIMITATIONS OF THE TESTS

Any diagnostic test should be judged by three fundamental criteria: (1) It should be fairly easy to do; (2) it should be constantly positive for the precise lesion concerned; and (3) it should be negative for all other lesions.

The grinding test is not too difficult, but it does need a good deal of practice and careful attention to detail. The use of a low couch, the application of considerable force in compression and rotation, and carrying out the test at the suitable angle of knee flexion are all important points, the neglect of which may result in failure.

As to whether the grinding test is always positive with a meniscus lesion and the distraction test with a soft-tissue lesion, it is perhaps too early to say. This paper is being presented at a somewhat early stage in the hope that more widespread trials will follow. Since becoming accustomed to the tests, however, the author has been surprised by their constancy.

With regard to other lesions, the author has three times found the grinding test positive when there was a pedunculated loose body other than a split meniscus. This does not seem to be a serious drawback for, quite apart from the help afforded by roentgenograms, operation was no less indicated.

RESULTS

The results by these methods are shown in Table I. The number of cases is small because, in the great majority of cases, the orthodox methods established the diagnosis simply and firmly. These cases are therefore omitted, and only fifty cases remain. Broadly speaking, Groups A and B are those in which only the application of the new tests permitted a diagnosis to be made; whereas Groups C and D, especially Group D, are those in which the tests resulted in the correction of wrong diagnoses.

Group A consists of ten cases. In none was there a history typical of meniscus injury, and in none was a single positive physical sign found by orthodox methods. In all ten, however, the grinding test was clearly positive, and in each a split meniscus was removed at operation.

TABLE I

RESULTS

Group	No. of Cases	Findings by Orthodox Methods			Findings by New Methods			Actual Diagnosis
		Typical History of Meniscus Injury	Positive Meniscus Tests	Diagnosis	Positive Grinding Test	Positive Distraction Test	Diagnosis	
A	10	0	0	No meniscus lesion	10	0	Split meniscus	Split meniscus at operation
B	13	Variable	0	Probably no meniscus lesion	13	0	Split meniscus	Split meniscus found at operation
C	15	Variable	0	? Split meniscus	0	15	Rotation sprain	Rotation sprain (not proved)
D	9	Fairly good	3	Probably a split meniscus	0	9	Rotation sprain	Rotation sprain
E	3	Miscellaneous cases for discussion						
Totals	50		3		23	24		

In the thirteen cases of *Group B*, orthodox physical examination was again negative; but the histories varied from complete vagueness to more or less definite hints of meniscus damage. Again, however, the grinding test was constantly positive; and in these cases, too, a split meniscus was removed at operation.

In a few of the cases in Group B, the history alone might possibly have led to operation. This links up with *Group C*, for in the fifteen cases of this group, there was a similar variability and vagueness in the history, coupled with absence of orthodox physical signs. To all appearances, therefore, Groups B and C are alike. In all the cases of Group C, however, the distraction test was positive, and a diagnosis of rotation sprain was therefore made.

Group D carries the argument a stage further, for it consists of nine cases in which the history definitely was suggestive of meniscus damage; moreover, in three of them a positive McMurray's sign was probably obtained. Again the grinding test was negative and the distraction test positive, and a rotation sprain was therefore diagnosed. With this diagnosis, in Groups C and D operation into the joint was not indicated. In a few cases negative air arthrograms were obtained, but the results with this method are insufficiently constant to afford proof. Purely conservative methods, including manipulation in some, appear to have justified the diagnosis. It is not unlikely that, without these tests, at least the three cases mentioned, and probably several more of this group, would have come to operation, and that normal menisci would have been removed. Here, then, are cases with an apparent diagnosis of meniscus damage, which were actually examples of rotation sprain. It is impossible to resist the speculation that this may provide the explanation for some of those enigmatic cases in which normal menisci are removed.

Group E consists of three miscellaneous cases. In one, the diagnosis of split meniscus was made on the basis of the history, a positive physical examination by orthodox methods, and also a positive grinding test; nevertheless a normal meniscus was removed. The remaining two cases had previously had meniscectomy; in both, a fragment of the posterior horn remained, diagnosed by the positive grinding test. They also showed a positive distraction test, however,—a curiosity explained by the presence of postoperative adhesions mimicking rotation sprain. Thus an apparent contradiction proved, in fact, to be confirmatory evidence of the validity of these tests.

One curious case was seen. The patient had a reasonably suggestive history and a very obvious McMurray's sign. When the grinding test was performed, and compression

and rotation were applied, there was a sudden click. Much to the patient's surprise, and somewhat to the author's, the knee was locked. To satisfy himself, the author locked and unlocked it three times in all. Fortunately this was not a very painful process; and it does seem to suggest that, in the grinding test, the correct type of force is being used.

SUMMARY

Some new tests for the diagnosis of meniscus injury, and its differentiation from rotation sprain, have been described. These tests aim at separating the individual components in knee-joint injury, and at reproducing the causal grinding force of meniscus damage. With wider experience, it is hoped that they may help to reduce the percentage of errors in the diagnosis of knee-joint injuries.

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MUSCLE FIBRODYSTROPHY

A Syndrome Causing Chronic Physical Disability

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The practice of orthopaedic surgery in the United States Army has afforded an unequalled opportunity for the examination of large numbers of young men. Deformities of the bones and joints and abnormalities of the neuromuscular system are frequently discovered by medical examining boards.

The majority of the patients examined and treated by orthopaedic surgeons have illnesses or injuries which clear up after short periods of rest, physical therapy, and general medical care. A very few have symptoms of more serious diseases or trauma, which require prolonged hospitalization and specific surgical treatment. In another group, the complaints arise from a residual disability from some injury or illness in civilian life. These latter patients are the most difficult to treat successfully in the Army. Most of them must be reclassified or discharged, under current military regulations. Among this group, members of the orthopaedic staffs of two Army Hospitals* in the Pacific Area have found an unusual number of patients with a characteristic neuromuscular syndrome, which constitutes a genuine, chronic physical disability. This condition does not seem to have been described or named previously.

Fibrodystrophy may be defined as a chronic, non-progressive muscle dystrophy, characterized by generalized weakness and contractures of all skeletal muscles. Most marked are the contractures of the erector spinae, the hamstrings, the calf muscles, and the extensors of the toes. Certain neuromuscular deficiencies of function appear as symptoms, such as poor coordination, inability to acquire skills which depend on fine muscle movements, and rapid exhaustion on physical exertion. Synonyms which could be used to describe this condition include "chronic myofibrosis", "chronic muscle contractures", "tightness of ligamentous structures", and "muscular fibrosis".

Onset of the Disease

In all of these cases the onset of the disease, when known, dated back many years. A majority of the patients were not aware of any physical handicap when they entered the Army; but, during physical training or subsequent combat duty, they found themselves lacking in physical stamina and strength. Thus the onset of symptoms and the recognition of their residual disability by most of these men occurred during the performance of duty with the Armed Forces.

Distribution of the Disease

During this study, large groups of men were examined in Army camps in Mississippi, Colorado, California, the South Pacific, the Philippine Islands, and Japan. They included soldiers from every state of the United States. In proportion to its population, no one part of the country contributed a larger number of cases of this syndrome than any other.

Incidence of the Syndrome

For this report, 264 cases of muscle fibrodystrophy have been studied and compared. These cases have been detected among approximately 12,000 patients, seen personally in orthopaedic clinics and wards of Army Hospitals during the past three and one-half years. A medical colleague, in an Evacuation Hospital in the South Pacific, has estimated that, among the 20,000 patients admitted to that Hospital in a two-year period, he had seen at

* The 31st General Hospital and 25th Evacuation Hospital.

least 200 additional cases of this condition. An incidence of 1 per cent. of the Army population is probably a conservative estimate. In a report by Kuhns, covering tightness of ligamentous structures in children, is a description of a similar condition occurring in 10 per cent. of a number of children whom he examined. It is possible that a survey of the civilian population would reveal an incidence of this syndrome of between 1 and 10 per cent.

Pathological Findings

The gross and microscopic pathology of this disease have not been fully demonstrated. In the Army, the opportunity was not given of doing a significant number of muscle biopsies. A few specimens which were examined suggest that there is an abnormal replacement of skeletal muscle fibers by connective tissue. One biopsy section was interpreted by the pathologist as "fibrosis and atrophy of skeletal muscle with fat and connective-tissue replacement". These sections were not diagnostic of a definite disease or of any single pathological process. No physiological or neuropathological investigation of the syndrome has been undertaken.

Clinical Description

The clinical picture of muscle fibrodystrophy is best illustrated by a typical patient.

A white soldier, twenty-two years old, in the fourth week of his basic training, was referred to the Orthopaedic Clinic for consultation. His chief complaint was severe pain in the muscles of his back and legs, occurring after a ten-mile hike, during which the patient carried a full infantry pack. For the three days since the march, these muscles had remained sore. The patient stated that he was unable to continue hikes or the usual morning calisthenics.

His past medical history showed no serious injuries, operations, or illnesses. At the age of sixteen he had been mildly ill for about three weeks in the summertime, with a condition which his family doctor diagnosed as "muscular rheumatism". He stated that during this sickness his muscles were "sore all over". He noticed considerable weakness when trying to walk, after getting out of bed for the first

FIG. 1

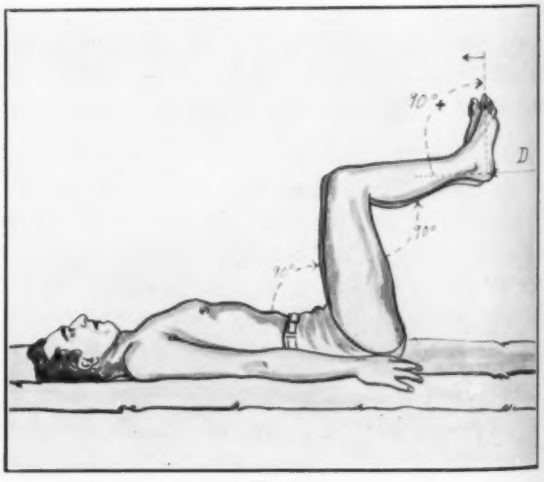

FIG. 2

Fig. 1: Contractures of erector spinae and hamstrings. Patient with fibrodystrophy is attempting the forward-bending test, trying to touch the floor with his finger tips while holding the knees straight. Note these characteristics:

A: Lumbar spine is flattened and forward flexibility is limited, due to contracture of the sacrospinalis.

B: Hamstring tendons are prominent; knees tend to flex before patient reaches his toes, due to contractures of hamstrings.

C: At extreme of physical effort, there remains a distance of ten inches or more from finger tips to floor.

Fig. 2: Contractures of calf muscles. In the ankle-dorsiflexion test, the patient lies supine with hips and knees at right angles, trying to dorsiflex his ankles.

D: Angle of dorsiflexion in cases of contracted calf muscles is 90 degrees or greater (combined effect of contractures of the gastrocnemius, soleus, posterior tibial, and peroneal muscles).

FIG. 3

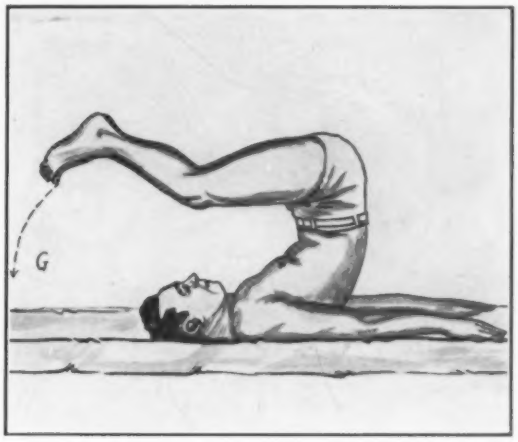

FIG. 4

Fig. 3: Contractures of hamstrings. In the hamstring test, the patient lies supine with hips held at a right angle, trying to extend his knees fully.

E: Angle of extension is 150 degrees, due to contracted hamstrings (combined effect of contractures of the biceps femoris, semitendinosus, and semimembranosus).

F: Note prominence of hamstring tendons at extreme of physical effort.

Fig. 4: Contractures of thoracic muscles. In the thoracic-muscle test, the patient tries to touch his toes to the floor above his head.

G: Note inability to reach the floor with the toes, due to some degree of contracture of all dorsal body muscles.

time. Later, he believed, he was never again quite so strong as he had been. While engaged in farm work, he would tire more quickly than other men on the same job. In high school he enrolled for a course in typewriting, but after three months he had to give it up because he could not attain the speed required. In athletics he tried out for football, but in spite of being a tall and heavy boy he made only the third team. He was clumsy in passing the ball and became quite exhausted after a short scrimmage.

On induction into the Army, he passed the required examinations and was given top classification for physical fitness and suitability for combat duty. Early in basic training, he found that calisthenics were more difficult for him than for other soldiers. On forward bending, he could not touch the ground with his finger tips. His feet and legs became very tired after a few minutes of running in position. When standing at attention, he found it difficult to keep his abdomen flat. He stated that he felt like a "farmer in uniform". He made short hikes easily; but, during long hikes and marches on paved roads and parade grounds, soreness developed in the muscles of his legs and thighs, and did not disappear after a night's rest. He had no complaints concerning his other body systems, and he seemed quite desirous of completing the course of basic military training with his friends.

Physical examination showed a well-developed, well-nourished young man in apparently good health. His height was six feet, weight 175 pounds. His posture showed a moderate increase of lumbar lordosis and a prominent and relaxed abdomen. The gait was good except for eversion of the feet of about 20 degrees, due to tight calf musculature. Laterally, his spine was straight. The small toes on both feet cocked up and barely touched the floor when he was standing. Examination of the head, neck, chest, and abdomen disclosed no other abnormalities. Examination of the muscular system showed that, while his skeletal muscles appeared normal in size and contour, they seemed softer than normal and lacked normal muscle tone. In testing his joints for range of motion, it was demonstrated that the apparent limitation was not due to any abnormality of the articulations of the spine and extremities themselves; but the restriction was in the lengths to which his muscles could be stretched.

Demonstration of Muscle Contractures

1. In bending forward, while standing with knees extended, the patient's finger tips could not reach closer than ten inches from the floor (Fig. 1). In this position, flatness of the lumbar spine was noted, due to contractures of the sacrospinalis muscles, which limited forward flexibility of the lumbar joints like "checkreins". At the extreme limit of bending, his knees would flex slightly and the hamstring tendons would appear prominent, due to contractures of the muscle bodies of the hamstrings.

2. When lying on his back, dorsiflexion of the feet at the ankles was limited to 90 degrees, regardless of whether his knees were flexed or completely extended (Fig. 2). This tightness of the calf muscles appeared to be due to contractures of both the gastrocnemius and soleus. On plantar flexion of the ankles, the toes assumed a cock-up position, due to contractures of the long extensor muscles of the toes. Joints of the feet and of the toes were very flexible, and there were no bone or joint deformities.

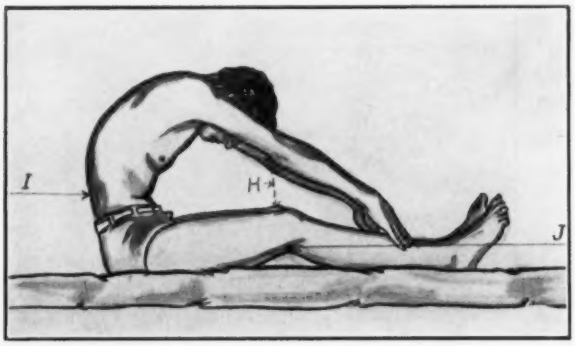

FIG. 5

Contractures of erector spinae. In the trunk-flexion test, the patient attempts to touch his head to his knees from a sitting position.

H: Note distance of ten inches or more from forehead to patellae.

I: Lumbar spine is flattened as a result of contracted sacrospinalis muscles.

J: Hamstring tendons are prominent as knees tend to flex at extreme of physical effort.

3. In attempting the straight leg-raising test, his knees could not be extended to more than 165 degrees. This was due to the bilateral contractures of the hamstrings (Fig. 3).

4. Contractures of all thoracic muscle groups were further shown by having the patient attempt to touch his toes to the floor above his head, while lying on his back (Fig. 4).

5. In a sitting position, bending forward, he could not bring his fingers closer than ten inches from his toes, and his knees would flex upon further effort. In trying to touch his head to his extended knees, he could not come closer than a distance of about ten inches (Fig. 5).

6. Palpation of the muscle groups in which the patient complained of pain showed that they were tender and slightly swollen at the junction of the muscular and tendinous portions. The sacrospinalis muscles were tender over their insertion on the sacrum.

The findings on neurological examination were within normal limits. All muscle and tendon reflexes were present, but slightly diminished. The Babinski and Romberg signs were absent. There was no spasticity or clonus. Temperature, pulse, respiration, and blood pressure were normal. Roentgenographic examinations of the lumbar spine and the feet showed no abnormality or congenital anomaly.

The clinical diagnosis was moderate muscle fibrodystrophy, caused by an undiagnosed illness at the age of sixteen.

CLINICAL FINDINGS

Variations in the Disease

Patients were referred to the orthopaedic consultant with a variety of symptoms and diagnoses. The clinical impression of the referring physician varied from "arthritis" and "neurosis" to "acute polyarticular rheumatism" and "malingering". "Myositis", "fibrositis", "strains" and "sprains" of muscles and joints, "tenosynovitis", and "psychoneurosis" were also frequent diagnoses. The symptoms, however, showed very few clinical variations. The patients most often stated that their symptoms had been brought on only by severe and unusual physical exertion. They complained of muscle soreness, tenderness, and pain on stretching their fatigued muscles. These symptoms were relieved by rest, heat, and massage; on return to duty—no matter how much they exercised—their muscles still remained weak and they would have recurrences of their symptoms after marches, hikes, obstacle courses, and similar training procedures.

Medical History

The medical histories of these patients are of unusual importance, as they indicate the probable etiology of this syndrome. Of the 264 patients included in this study, 112 dated their disability from some definite illness in childhood. Forty-eight of these stated that this illness was a recognized attack of "infantile paralysis" (Table I). From their own memories or from information received from other members of their families, a few could give further details. Twenty of these patients had been hospitalized for infantile paralysis and discharged as "cured", after from three weeks to six months of treatment. Twenty-eight others were treated at home by their family physicians, who in most cases had stated that the patient had fully recovered from a mild case of poliomyelitis, without residual paralysis or deformity. Sixty-four others gave a positive history of some episode in childhood which closely resembled a mild attack of poliomyelitis. These illnesses were

TABLE I

PAST MEDICAL HISTORY

	Number of Patients
Known diagnosis of poliomyelitis	48
Illness resembling poliomyelitis	64
No known causative illness	152
Total	264

variously described as "epidemic fever", "paralysis fever", "flu in the throat", "leg-muscle paralysis", "summer fever", "muscular rheumatism", "diphtheria paralysis", the "flu", and the "grippe". Several of the patients described symptoms which are common in mild attacks of poliomyelitis,—such as, a preceding infection in the upper respiratory tract, gastro-intestinal upset and vomiting, fever, sore throat, headache, and weakness in swallowing, simulating a bulbar type of poliomyelitis. Almost all described muscle pain and tenderness, stiffness of the neck, and stiffness and soreness in the muscles of the legs and back. Many stated that they were weak for several months after their illness. Some of them reported difficulty in learning to walk again, or had an unsteady gait after the illness. On questioning, those who were afflicted in their teens often knew of other cases of infantile paralysis in the same neighborhood during the summer months in which they were ill. At the time, however, they did not realize that their illness might have been a mild form of the same disease. Eight patients stated that some other members of their family had been ill the same year with a paralytic form of poliomyelitis, including residual muscle weakness and atrophy. Only nine were sure that the diagnosis of poliomyelitis had been confirmed by spinal-fluid examination. Six had been in casts or splints during the acute stage of the illness. Three patients had worn braces during the convalescent period. In one patient a stabilizing operation of the right foot had been performed.

One hundred and fifty-two patients in this series could give no definite history of any illness resembling poliomyelitis. Some of them had had severe illnesses in infancy or early childhood, in which a definite diagnosis was not remembered by the patient. In spite of the negative medical histories of the majority of these patients, there was no detectable difference in the presenting symptoms and physical findings of those who knew that they had had poliomyelitis and those who did not.

Careful comparison of the physical prowess and athletic ability of these patients indicated many deficiencies. They had never been physically strong, as compared with other boys of similar ages. They had noticed unusual fatigue and exhaustion, and previously they had attributed these symptoms to being "naturally weak" or "short of wind". If this condition had been recognized early in their lives, they had accepted it naturally and had adjusted their physical activities to their disability, without being aware of the cause or full extent of their handicap. Most of them had been in sedentary trades or occupations not requiring prolonged physical exertion. Only a few had been farmers. Most of them were students, clerks, salesmen, or office workers. In school they had not excelled in any athletic activity. If they engaged in competitive sports, they had failed to make the team. Some of them were fair in tennis, swimming, or golf, but not on a competitive level. In addition to rapid fatigue and exhaustion, they had noticed poor coordination, that they were weaker than other men, and that their muscles had not developed in size or power.

Lack of coordination and fine muscle movement was also evidenced by poor handwriting. Not a single patient had acquired the ability to play well any musical instruments which require finger coordination,—such as, the piano, stringed instruments, or reed instruments. In contrast to their physical accomplishments, they usually showed a high record of intelligence and scholastic performance in school and college. Most of them had been successful in business and social life. One fact found in every history was the lack of the

TABLE II
BODY TYPES

	Number of Patients
Asthenic habitus	168
Obese	80
"Muscle-bound"	16
Total	264

progressive nature of this physical disability. On this, the patients were quite definite. This is a characteristic and diagnostic feature of the syndrome.

In the Army, the majority of these patients failed to complete basic infantry training. Almost all of them had to be reclassified for limited duty because of muscle fibrodystrophy. It seemed apparent, however, that these patients had made more than an ordinary effort to complete their physical training, even at the expenditure of unusual amounts of physical and nervous energy; and they had endured for some time considerable pain, fatigue, and discomfort.

Physical Findings

Physical examinations of these patients showed three types of body conformation and development. The majority of the patients comprised a group having asthenic habitus and poor muscle development. They tended to be thin, stooped, and slightly underweight; they had increased lumbar lordosis and prominent abdomens. The patients in the second group were heavier in build and weight, but showed abnormal depositions of fat. The smallest group of patients showed good muscle development, but they were markedly "muscle-bound" (Table II).

Postural and static deformities were common (Table III). The spine showed an increased thoracic curve, increased lumbar lordosis, and scoliosis, with compensated symmetrical S curves to both right and left and mild rotation. The pelvis was frequently tilted forward, but no obliquities were noted. Slight to moderate recurvatum was the most frequent deformity noted in the knees, and there were several cases of slight to moderate bowing of the tibiae. The deformity most frequently seen in the foot was cocking up of the smaller toes. Pes planus and pes cavus were found in about equal numbers; they were associated with contracted peroneal muscles or with contracted plantar fascia and short flexor muscles of the toes, respectively. About 5 per cent. of the patients showed unilateral asymmetry of the lower extremities. Slight to moderate atrophy of the thigh and calf muscles frequently was present. Occasionally there was a measurable weakness in one or

TABLE III
DEFORMITIES

	No. of Deformities	Number of Patients
No deformities		84
Mild deformities		180
Scoliosis	22	
Round shoulders	46	
Exaggerated lordosis	60	
Pelvic obliquity	0	
Unequal leg lengths	11	
Unilateral muscle atrophy	21	
Genu recurvatum	3	
Pes planus	18	
Pes cavus	33	
Cock-up of toes	26	
Miscellaneous	4	
Total	244	264

TABLE IV
MUSCLE CONTRACTURES IN 264 PATIENTS

Muscle	Number of Patients
Sacrospinalis	264
Hamstrings	264
Calf muscles	140
Plantar fascia	30
Toe extensors	23
Upper trapezius	6
Pectoral muscles	5
Psoas muscles	3
Miscellaneous	5

the other leg, chiefly in the anterior peroneal or quadriceps muscles. Several of the patients showed shortening of the weaker lower extremity of from one-half inch to an inch. No cases of weakness or atrophy of the abdominal or trunk muscles, or of the upper extremities, were noted.

The one positive physical finding common to all of these patients was contracture and shortening of the sacrospinalis and hamstring muscles (Table IV). No cases were found which had shortening in one of these groups without having contractures in the other, also. Most of the patients, however, had more extensive involvement than simply these two muscle groups. Contractures of the calf muscles, plantar fascia, extensor muscles to the toes, peronaei, tibialis posterior, hip adductors, longissimus dorsi, and pectoralis major muscles were also frequent findings. Wherever tests for poor muscle development and contractures of the spine and hamstrings were positive, a typical history of previous physical handicaps and of other symptoms of muscle fibrodystrophy, confirming this diagnosis, could be obtained. Neurological examinations of these patients showed no single typical picture. Sensory examinations and the cranial-nerve examinations were always normal, but the reflex response and the degree of muscle atrophy were extremely varied. The most common finding was that of diminished tendon reflexes, particularly in the lower extremities. Tendon reflexes were absent in a few patients. Rarely, patients were found to have hyperreflexia, with unsustained ankle or patellar clonus. The abdominal and cremasteric reflexes were normal. The Babinski sign was absent in all cases.

The laboratory findings, in the forty-two patients who were hospitalized for study, were all within normal limits. The serology was negative. The red blood counts and hemoglobin were normal. The white blood counts were not elevated. There was no eosinophilia. Urinalyses were normal. Roentgenograms of the chest were normal. Sedimentation rates were not elevated. Malaria smears from a dozen patients were negative. Lumbar punctures on eight patients showed normal cell counts and protein in the spinal fluid. There was no evidence of circulatory block or of increased pressure due to cerebrospinal fluid. Roentgenograms of the bones and joints in twenty of the patients showed one congenital anomaly of the lumbosacral joint. There were four cases of well-compensated scoliosis. Two of the patients in the hospital had enlarged and chronically infected tonsils. In several patients infected teeth required removal. The incidence of foci of infection was very small, and seemed in no way related to the patient's symptoms of muscle pain.

Differential Diagnosis

The findings in the 264 patients in this series diagnosed as having muscle fibrodystrophy resemble most closely the clinical picture of patients who have recovered from paralytic attacks of acute anterior poliomyelitis. However, generalized muscle contractures are the main features of fibrodystrophy. In paralytic poliomyelitis, the most notable features are the weakness and muscle atrophy which usually follow the paralysis.

Pathological types of muscle contracture are uncommon in the medical literature. Kuhns has reported a congenital type of muscle contracture which he designates as "tight-

ness of ligamentous structures". He found this condition in approximately 10 per cent. of the children examined. Easy fatigability and pain in the back after hard playing were common findings. A familial incidence was present in more than one-half of the cases, and minor developmental deformities of the lower part of the back were found by roentgenographic examination in one-quarter of his patients. Kuhns believes that the contracture is congenital in origin, but there may be acquired forms as a result of prolonged fixation in bed, casts and braces, and vitamin-C deficiency.

The possibility that these cases of fibrodystrophy might represent some form of myositis or chronic infectious myopathy has been considered. At the time of examination, none of the patients showed evidence of any acute or chronic inflammatory or infectious disease. In the involved muscles there was no sign of local inflammation or tender nodules, such as are usually found in myositis or fibrositis. Fibrodystrophy can be differentiated from rheumatism and rheumatoid arthritis by the normal sedimentation rate and the complete absence of joint phenomena, periarticular swelling and tenderness, and arthritic involvement of the joint surfaces. Roentgenographic studies of the joints in fibrodystrophy disclose no pathological changes which are characteristic of, or can be associated with, the disease process.

Chronic intramuscular fibrositis in the residual stages, as described by Slocumb, resembles somewhat the picture of muscle fibrodystrophy. In intramuscular fibrositis, however, an active disease process is present; and the symptoms are worse after periods of inactivity than after exertion, as in fibrodystrophy. Patients with intramuscular fibrositis have definite stiffness in the morning and aching pains related to changes of temperature and humidity. The patients reported here had mildly aggravated symptoms in cold weather, but they noticed no increase in fatigue or muscle pains due to dampness, and morning stiffness was not a characteristic symptom.

The myopathies and the hereditary and familial diseases of the neuromuscular system have been ruled out in the differential diagnosis of muscle fibrodystrophy. This syndrome is not progressive in character; the onset may occur at any period during infancy or childhood; spasticity and hypertrophy of the muscle bodies are completely absent; abnormal reflexes are not present; and there is complete freedom of involvement of the cranial nerves and of the functions of the cerebrum and cerebellum.

Other chronic diseases of the neuromuscular system have been ruled out by similar comparisons. In chronic degenerative myelitis, sensory abnormalities are always present; and a loss of sphincter control usually occurs in the advanced stages. In combined and multiple sclerosis there are characteristic disturbances of gait; Babinski and Romberg signs are present; and there is definite spasticity of the muscles and extremities.

Prognosis

A number of these patients were followed for as long as three years in the Military Service. During this time, none of them showed any progression in the signs and symptoms of the disease. Some, who had been aware of their physical disabilities for ten years or more, stated that they had noticed no increase in muscle weakness, stiffness, or other changes, which could not be attributed to their advancing age. Since this syndrome is a residual physical disability, rather than an active disease process, the prognosis for these patients is excellent.

TREATMENT

In this series of patients, treatment was necessarily limited to their presenting symptoms. These were, in order of frequency, pain in the muscles of the back and legs, tenderness in the muscles on stretching of the musculotendinous junction, exhaustion and fatigue, and a feeling of muscle weakness and loss of strength. The mild cases were

treated by physical therapy,—principally infra-red rays and massage; aspirin was given for its analgesic action; and a request was sent to the patient's military unit, recommending assignment to light duty for a period of from five to ten days, or until the muscle symptoms disappeared. In the more severe cases, the men were admitted to the hospital, where they were put on the regimen of bed rest and intensive physical therapy,—principally whirlpool baths, followed by massage, and active exercises for improving posture and strengthening individual muscle groups. These patients were usually treated for from one to three weeks. Those in whom the syndrome seemed so pronounced as to interfere with their military duties were recommended for reassignment to other units, or to jobs in the same unit which did not require so much marching, lifting, or carrying of heavy packs. A total of seventy-two patients were reassigned to limited service. No patient had to be separated from the Army because of this condition.

PSYCHOLOGICAL FACTORS

As with the majority of patients in the Military Service, attention had to be given to the mental aspects of this condition,—as regards both symptoms and treatment. A large number were referred to the orthopaedic clinics from the Neuropsychiatric Section of the Medical Service, where they had been admitted for study and observation. Some had already been given various neurological or psychiatric diagnoses. "Neurasthenia", "effort syndrome", "unclassified psychoneurosis", "hysteria", "anxiety state", "suspected malingering", and "constitutional psychopathology" were the most frequent functional diagnoses applied to this syndrome. There was justification for the development of a mild psychoneurosis in some of these patients. They became aware that they had a disease condition which produced definite symptoms, but in the diagnosis of which the doctors could not concur, explain in terms which the patient could understand, or treat so that the symptoms would not return. When first examined, many patients were tense, nervous, worried about their physical state, apprehensive about the possible progress of the disorder, and resentful of the fact they were considered "mental patients", when the symptoms in the beginning had been only in their muscles. The best psychological treatment was to explain to the patient in simple terms the true cause and nature of his disability. With the more intelligent patient, the examiner would point out that the condition was not a "disease", but was merely the result of an infection in childhood,—probably a mild attack of poliomyelitis. The patient would be reassured that it was not serious, not progressive, and not in any way a threat to future health and earning power upon return to civilian life. It was demonstrated that the symptoms could be relieved by short periods of physical therapy, and that they could be avoided by keeping physical exertions within the limits of strength and endurance. Exercises were recommended and sports were suggested in which the patient would not be so apt to strain himself,—that is, swimming, horseback riding, golf, and daily calisthenics. These patients were encouraged to build up their general physical strength and endurance by regular periods of active exercise; but they were advised to rest before their symptoms became noticeable. The less intelligent soldier was told that his symptoms were due to the fact that he was "muscle-bound". He was assured that it was not a serious condition; that it would never be any worse; and that, by being careful not to overexert himself, he could lead an active life without recurrence of his symptoms.

The results from treatment—both physical therapy and this type of psychotherapy—were excellent. In fact, the effects of treatment were so good as to constitute a valuable differential test between this syndrome and the purely psychoneurotic disorders. The patients with muscle fibrodystrophy responded rapidly and completely to treatment; they noticed definite improvement with each day of physical therapy; and in from one to three weeks they were ready to return to duty. The psychoneurotic patients, on the other hand, were usually relieved only temporarily, or not at all, by physical therapy. Moreover, when a

psychoneurotic patient was ordered "to duty", it was very common to note an acute "recurrence" of all his previous symptoms.

ETIOLOGY

In the medical histories of this series of patients, there was such a high incidence of acute anterior poliomyelitis that the author has concluded that poliomyelitis is the most important cause of this syndrome. A careful comparison of cases in which there was no definite history of poliomyelitis showed that the symptoms and physical findings were identical with those of patients who had a positive history of poliomyelitis. This suggests the hypothesis that acute anterior poliomyelitis is the *only* cause of muscle fibrodystrophy.

This theory is supported by the observations of other workers, reported within the past few years, on the high incidence of non-paralytic forms of poliomyelitis. Sherman, in a review of the medical literature and a report of seventy cases, has emphasized that only 10 per cent. of her patients with acute anterior poliomyelitis had sufficient residual weakness to require braces or future surgery; 8.6 per cent. had functional weakness, which did not require further therapy and was not a handicap to normal life; and 72.8 per cent. either had no residual weakness or such slight weakness that it was barely detectable (8.6 per cent. of the patients died). However, her patients were not followed for a sufficient length of time to determine the incidence of deformities or muscle contractures which developed during the growth period.

Swartout and Frank, by using the presence of muscle spasms as a new criterion for the diagnosis of acute anterior poliomyelitis by mild physical findings, were able to detect multiple familial cases of poliomyelitis which would otherwise not have been diagnosed. They reported twenty-nine families with multiple cases of infantile paralysis, many of them so mild that, if it had not been for at least one severe case in the family, the other cases would not have been correctly diagnosed. They further state: "The lack of recognition of these mild cases may account in some measure for the difference between the large number of adults who have protective substances in their blood against poliomyelitis virus and the comparatively small number who have a history of having had the disease. Unrecognized, and therefore untreated, one-sided spasm may account for the frequent orthopaedic problems of unexplained scoliosis and leg shortening."

Electromyographic studies by many workers within recent years have demonstrated that muscle spasm in acute anterior poliomyelitis is a more extensive symptom than muscle weakness and paralysis. It is inevitably present in the muscles of the lumbar spine and the hamstrings, and frequently it is present in the muscles of the upper and lower extremities. Schwartz, Bouman, and Smith have emphasized that spasticity is a general phenomenon in the early stages of infantile paralysis. By their studies, they have emphasized the general systemic effect of this disease on the muscle system.

Nielsen, in 1944, reported four cases of a generalized subacute exhaustion syndrome of the neuromuscular system, which the author believes could have been due to atypical attacks of acute anterior poliomyelitis in adults who were physically exhausted at the time of onset. His cases differ from these considered here in that their onset was in adult life and was associated with considerable muscle atrophy.

Many patients have been examined by the author, who showed some degree of muscle spasm resulting from diseases other than infantile paralysis, but it is significant that in none of them did muscle contractures or fibrosis develop subsequently. Six patients with encephalitis in an overseas Army Hospital all had, temporarily, stiff necks and spasm of the spine muscles during the acute course of the disease. Over one hundred cases of meningococcal meningitis were seen in an epidemic in an Army camp in this country. Transient muscle spasm was found in the neck, spine, and hamstrings, and frequently in the hip and shoulder muscles. Temporary atrophy was the rule with these patients, yet no permanent muscle contractures or fibrosis was seen after a follow-up of three months.

More or less involvement by muscle spasm, during some stage of the acute attack, occurs in rheumatism, rheumatoid arthritis, myofibrositis, and infectious polyneuritis. Periarticular fibrositis, with limitation of joint motion, occurs in rheumatoid arthritis; but this is quite different from the limitation of joint motion after poliomyelitis. In poliomyelitis the limitation is due to muscle spasm, and finally to interstitial fibrosis in the muscle body. The joints themselves and the joint capsules are seldom involved, and they more frequently show relaxation and hypermobility than contractures and fibrosis.

New information on the extent and the contagious nature of poliomyelitis in children has been reported by Casey and his co-workers. They found that poliomyelitis was contagious in about 90 per cent. of children from one and one-half to three and one-half years of age. Multiple cases of poliomyelitis in one family were the rule rather than the exception, when there were other children from one and one-half to eight and one-half years of age in the same home. These authors investigated the immediate family and the neighborhood of seven paralytic cases of poliomyelitis. Of sixty-six persons in contact with these seven during the infectious period, illnesses which were compatible with poliomyelitis developed in thirty-seven, within six to fifteen days; and twenty-four persons were definitely considered to have poliomyelitis. This is a ratio of infectivity of sixty-one to sixty-six. In two control groups among 224 persons who were not in contact with poliomyelitic patients, illnesses compatible with poliomyelitis developed in only nine, and not one was a significant clinical case. This is further proof that the number of persons infected with poliomyelitis among the general population is much greater than clinicians have previously realized. It is the author's opinion that, among this large group of patients with mild, abortive, non-diagnosed, and non-paralytic forms of acute anterior poliomyelitis, the clinical syndrome of muscle fibrodystrophy develops in an unknown number, with subsequent lifelong physical disability and occupational handicaps.

Smith and her associates, in a study of the origin of an epidemic of poliomyelitis in Buffalo, have demonstrated that, preceding an epidemic of recognized cases of poliomyelitis with paralysis, there are in the community many minor illnesses which are non-paralytic forms of the disease. These are often not recognized by the patients, their families, or their physicians as poliomyelitis until the paralytic cases begin to appear. These investigators have estimated that the incidence of illnesses in the community, which were highly suggestive of being non-paralytic forms of the disease, was at least five times the number of recognized poliomyelitis cases. All gradations of symptoms and of severity were encountered, and as late as one year after the epidemic some patients had signs and symptoms—such as a degree of residual weakness, atrophy of one leg, and other deformities—which finally confirmed diagnoses of poliomyelitis in what otherwise seemed to be mild illnesses.

SUMMARY

Fibrodystrophy is the proposed name for a neuromuscular syndrome, found in a series of 264 patients from the United States Army. It was originally discovered as a cause for soldiers' complaints of excessive pain and fatigue in their muscles after moderate physical exertion.

Patients with this condition are characteristically lacking in athletic ability. They have great difficulty in acquiring skill in occupations requiring fine muscle coordination,—such as typewriting, playing musical instruments, and in rifle marksmanship.

Physical examinations show three body types,—"asthenic", "obese", and "muscle-bound". Five simple muscle tests demonstrate the common findings,—contractures of the dorsal musculature of the body, particularly of the erector spinae, the hamstrings, and the calf muscle groups. Mild deformities—such as scoliosis, pes planus, and dorsiflexion of the interphalangeal joints of the toes—are frequently found, along with moderate muscle weakness and occasional atrophy.

The syndrome is a cause of lifelong physical disability, but it does not seem to be progressive after the adolescent growth period. Individuals thus affected, on entering military service, frequently for the first time became aware of their poor muscle development, impaired coordination, and lack of physical endurance.

The etiology of fibrodystrophy is presumed to be abortive or non-paralytic attacks of acute anterior poliomyelitis during the patient's infancy or childhood. A significant number of patients in this series either had a recognized case of poliomyelitis in their youth or some illness clinically similar to the milder forms of this disease. The muscle contractures, which are the outstanding clinical and diagnostic features of the syndrome, are considered to be the residual pathological changes of interstitial muscle fibrosis and atrophy, following the untreated muscle spasm of the acute illness.

Treatment of this condition, consisting of rest and physical therapy, is successful in relieving the patients' symptoms of muscle pain and tenderness. For the permanent pathological changes in the muscles, no specific treatment is known. The soldiers in this series were reclassified for "limited service" and duty not requiring prolonged physical exertion.

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PSEUDARTHROSIS OF THE LONG BONES

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Non-union of fractures is not a new subject. However, it is easy to associate the occurrence of fractures to some extent with modern living, in which serious injuries frequently occur in civil as well as in military life. It is perhaps more appropriate to relate non-union to some of the newer ideas concerning the treatment of fractures. Many surgeons of the past century, like Malgaigne and Bardenheuer, never saw pseudarthrosis in closed fractures of the shafts of the bones, because they reduced the fractures accurately and kept the bones immobilized long enough to secure healing.

In accordance with Bruns's statistics, quoted by Böhler, pseudarthrosis occurs once in 200 or 250 cases, or in 0.5 per cent. of all diaphyseal fractures; according to Scudder, it occurs in from 2 to 3 per cent.; and according to Hey Groves, in from 4 to 5 per cent. Bado, in his thesis in 1941, says that pseudarthrosis is a very rare condition; and, for its occurrence, it is necessary that a strong and persistent foreign factor, opposed to the natural tendency of bone healing, be present. This factor, according to Bado, is almost always an incorrect method of treatment. Watson-Jones considers that: "Non-union of fractures is due to the failure of surgeons much more than to the failure of osteoblasts". Of course, pseudarthrosis is not a disease. It is normal for a fractured bone to heal, and non-union can almost always be avoided. In a series of 800 consecutive fractures of the shaft of the femur or tibia recently investigated by Watson-Jones, all types of treatment were used. A high percentage of these fractures were comminuted, contaminated, and severely infected. Because good orthopaedic methods were used, there was not one case of non-union in the entire series, although many fractures were slow in uniting. Ottolenghi from Buenos Aires, in a series of 172 fractures of the shaft of the humerus, reports only thirteen cases of non-union; and specifies that, of these cases, twelve were treated elsewhere, indicating that he had only one case of non-union in the series. Open reduction was used only eight times.

The present paper is based upon work done at the Orthopaedic Department of the State University Hospitals, Iowa City. Since most of the original fractures were treated elsewhere, it is not possible to give percentages of non-union in relation to the number of fractures treated. It is possible, however, to be analytical, and to review and institute

TABLE I

DISTRIBUTION OF CASES

Bones	Females		Males		Total	
	No. of Cases	Per Cent.	No. of Cases	Per Cent.	No. of Cases	Per Cent.
Femur	3	2.2	18	13.4	21	15.6
Tibia	16	11.9	32	23.7	48	35.6
Tibia and fibula	2	1.5	4	2.9	6	4.4
Humerus	12	8.9	6	4.5	18	13.4
Radius	1	0.7	6	4.5	7	5.2
Ulna	1	0.7	7	5.2	8	5.9
Radius and ulna	3	2.2	19	14.0	22	16.2
Clavicle	3	2.2	2	1.5	5	3.7
Totals	41	30.3	94	69.7	135	100.0

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TABLE II

DEVELOPMENT OF PSEUDARTHROSIS

Ages (*Years*)	Number of Cases	Per Cent.
5 to 10	5	3.7
11 to 20	16	11.9
21 to 30	26	19.3
31 to 40	25	18.5
41 to 50	25	18.5
51 to 60	15	11.1
61 to 70	18	13.3
Over 70	5	3.7
Totals	135	100.0

details of treatment which might decrease the incidence of this disconcerting complication. Of 346 cases of pseudarthrosis seen in this Department from 1935 to 1945, 391 bones were involved. One hundred and thirty-five cases of non-union involving the long bones were selected, excluding cases of pseudarthrosis of the femoral neck, which alone numbered 128. The 135 cases mentioned form the basis of the present paper. Attention was focused only on cases which had the characteristic signs of non-union, clinically or roentgenographically,—namely, motion *in situ* or sclerosis of the bone ends. In accordance with these criteria, some cases should have been included which had motion at the fracture site, but they were thought to be merely cases of delayed union. It was believed that in these cases the bones would heal if immobilized long enough, and consolidation did occur in many of the cases handled in this manner. They are not included in this paper.

Table I indicates the distribution of cases according to sex and location. These figures cannot be used as a criterion for the frequency and distribution in the general population, but only as an indication of the frequency in the present series. The author feels that these ratios are related to the activities and occupations of the two groups.

The ages at which pseudarthrosis developed in this series are shown in Table II. The youngest patient was five years old and the oldest was eighty-four.

The relationship between the pseudarthrosis and the type of fracture was analyzed. Table III points out the number of compound and simple fractures in which non-union developed. The frequency of occurrence of non-union in simple fractures may be explained by the fact that the number of simple fractures far exceeds that of compound fractures, and also that generally the treatment of the simple fractures was more neglected.

TABLE III

OCCURRENCE OF NON-UNION

Bones	Compound Fractures		Simple Fractures	
	No. of Cases	Per Cent.	No. of Cases	Per Cent.
Femur	8	5.9	13	9.6
Tibia and fibula	27	20.0	27	20.0
Humerus	5	3.7	13	9.6
Radius	0	0	7	5.3
Ulna	3	2.2	5	3.7
Radius and ulna	8	5.9	14	10.4
Clavicle	0	0	5	3.7
Totals	51	37.7	84	62.3

The causes of non-union, as reported by many authors, are the following: (1) gross separation of the fragments; (2) interposition of soft tissues; (3) improper immobilization; (4) damage of the nutrient artery; (5) inadequacy of blood minerals, like calcium and phosphorus, or deficiency of vitamins C and D; and (6) pathological conditions of the bone, such as carcinoma or Paget's disease. In the light of present knowledge, it is practically impossible to consider one factor alone as the cause of non-union. It is evident that in some cases there is inadequate consolidation, but the etiology of this abnormality is difficult to ascertain. However, the surgeon can aid consolidation by (1) efficient apposition of the fragments, (2) adequate and uninterrupted immobilization, (3) prolonged immobilization, and (4) the avoidance of repeated attempts at reduction. Some features of this subject are surprising and inexplicable. For instance, we see many robust, healthy individuals with fractures which would be expected to heal without incident; yet pseudarthrosis is obtained. On the other hand, we see cases of osteogenesis imperfecta with multiple fractures which heal well. Steindler quotes Muscatello and Damarchelli, who in 1899 found that, even after section of the peripheral nerves, there was no disturbance in the formation of callus, and consolidation occurred without complication.

Inadequate circulation is often mentioned in the literature as a major factor in the production of non-union. It is easy to understand that an inadequate supply of blood can be of paramount importance in the healing of fractures of the femoral neck or of the carpal navicular; but the explanation is not so clear in fractures of the shafts of the long bones, since the circulation is assured by the rich blood supply of (1) the nutrient artery, (2) the periosteal arteries of the shaft, and (3) the periosteal arteries of the epiphysis. The nutrient artery divides in the marrow into two branches, one going upward and one downward. The main branch of the nutrient artery follows the direction of the nutrient foramen; and it will course distally in the humerus, proximally in the femur, and so forth. Both branches of the nutrient artery give off smaller branches for the nutrition of the marrow and those which penetrate the haversian canals and then anastomose with the arterial ramifications which come from the periosteal arteries. When the branches of the nutrient artery terminate in the epiphysis, they anastomose with the ramifications that the epiphysis receives from the periosteum. The periosteal arteries of the shaft arise from the periosteum. They are present in large numbers and penetrate the bone through third-order foramina as capillaries within the haversian system, anastomosing as already described. The periosteal arteries of the epiphyses originate from the periosteal layer which lines the epiphysis, and penetrate through second-order and third-order foramina. This would indicate that the three systems of arteries having such good anastomoses provide adequate circulation for bone nutrition, even in cases where there might be some interruption of any of these systems.

In Table IV are recorded the locations of the fractures of the long bones. Of the 135

TABLE IV

LOCATIONS OF FRACTURES OF THE LONG BONES

Bone	Total Number	Upper Third		Junction of Upper and Middle Thirds		Middle Third		Junction of Middle and Lower Thirds		Lower Third	
		No.	Per Cent.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Femur	21	6	28.6	0	0	9	42.8	3	14.3	3	14.3
Tibia	54	11	20.4	4	7.4	17	31.4	11	20.4	11	20.4
Humerus	18	2	11.1	1	5.5	5	27.8	3	16.7	7	38.9
Ulna	24	4	16.7	4	16.7	0	0	12	50.0	4	16.7
Radius	25	2	8.0	1	4.0	12	48.0	5	20.0	5	20.0
Totals	142	25		10		43		34		30	

TABLE V

PATHOLOGICAL FINDINGS

Pathological Findings	No. of Cases	Surgery	Good Result	Failure
Diabetes	3	2	2	0
Decreased phosphatase	1	1	1	0
Obesity (250 pounds)	1	1	1	0
Positive Wassermann and Kline tests	4	3	2	1
Paget's disease	1	0		
Epilepsy	1	1	1	0
Tabes and other diseases of central nervous system	2	2	1	1
Arteriosclerosis	2	2	1	1
Moon-Biedl syndrome	1	1	1	0
Increased non-protein nitrogen	1	1	1	0
Totals	17	14	11	3

cases studied, the site of the pseudarthrosis was specified in 142 bones. The table shows that, in the femur, most of the cases of pseudarthrosis were located in the middle third (42.8 per cent.); the second most frequent site was the upper third (28.6 per cent.). In the tibia, also, most of the sites were in the middle third. However, it is evident that the frequency is predominantly in the lower half of this bone. In the humerus, and also in the radius and ulna, pseudarthrosis occurred most frequently in the section including the junction between the middle and lower thirds and the lower third.

In this series, most of the patients with pseudarthrosis had normal levels of blood calcium, phosphorus, and phosphatase. In seventeen cases some pathological finding was present (Table V). The abnormalities in these cases probably had little relation to the production of the non-union. Fourteen of these patients were operated upon, with only three failures.

Some authors feel that the healing of one bone in the forearm or leg may be retarded by the other intact, or more rapidly healing, bone, which acts as a splint and keeps the fragments separated. This belief is substantiated by the following data, obtained in this series:

1. In thirty-two of fifty-eight cases of non-union of the tibia (55.2 per cent.), the fibula was solid.

2. In five of thirty-seven cases of non-union of the radius (13.5 per cent.), the ulna was solid.

3. In eight of thirty-seven cases of non-union of the ulna (21.6 per cent.), the radius was solid.

Fifty cases of this series had been treated elsewhere by open reduction, skeletal traction, débridement, sequestrectomy, wiring, plating, metal bands, or other forms of external

TABLE VI

MASSIVE ONLAY BONE GRAFT WITH VITALLIUM SCREWS

Bones	Number of Cases	Results	
		Good	Poor
Femur	13	11	2
Tibia	13	12	1
Humerus	4	3	1
Radius	2	2	0
Ulna	3	3	0
Radius and ulna	14	12	2
Clavicle	2	2	0
Totals	51	45 (88.2%)	6 (11.8%)

TABLE VII

MASSIVE DUAL ONLAY GRAFT WITH VITALLIUM SCREWS

Bones	Number of Cases	Results	
		Good	Poor
Femur	1	1	0
Tibia	4	4	0
Humerus	3	2	1
Totals	8	7 (87.5%)	1

fixation. Some of the patients were seen after treatment, which consisted of bone grafts, bands, or drilling procedures. In a considerable number of cases of fractures of the femur in which pseudarthrosis developed, traction had been applied. Thirteen patients out of twenty-one (61.9 per cent.) had had this type of treatment. In non-union of the tibia, there was also a high incidence of such treatment. In one case of non-union of the ulna, the patient had been exposed to thirteen general anaesthetics and had had seven open reductions! Flagrant violations of adequate immobilization occurred in two cases, in one of which a fracture of the forearm had been treated with plaster immobilization only from the wrist to the elbow; in the other, a fracture of the tibia had been immobilized merely by a cast from the ankle to just above the knee. In addition, many of the patients had been subjected to several changes of cast within relatively short periods of time.

TREATMENT

The only treatment for non-union is surgical. If, however, the orthopaedic surgeon will wait and observe, he will find that many of the cases of so-called non-union are truly cases of delayed union, which will heal with proper immobilization and time. When clinically there is motion and when roentgenographically there is occlusion of the bone ends because of sclerosis, we may say that pseudarthrosis has already taken place. Surgery is then the only solution.

Many varied surgical procedures are being used, some of which have been accepted by their various proponents as adequate. Of these, Beck's perforations—used by Böhler since 1929, but described previously by Wildey in 1914—and the use of bands and pins are being replaced today, because of their less favorable results as compared with the more modern treatment by bone-grafting. The grafting of the affected bone was first recommended by Wolff and Ollier, and then performed by Nussbaum in 1860. When bone grafts are used, the same principles should be observed as in good fracture treatment. Bado states that all bone-graft operations will be successful if the surgeons follow the principles used in the treatment of fractures. Inattention to these principles will result in reproduction of the identical conditions which contributed to the non-union. There is no essential difference between the problems of bone healing after fracture and after bone-grafting. In the former case, the problem is one of healing following accidental trauma, whereas in bone-grafting it is one of healing following surgical trauma. The principles of bone-grafting, when correctly applied, will give good results no matter what method is used.

Various kinds of bone grafts have been used by different orthopaedic surgeons, but today there is almost a unanimous tendency to use only autogenous grafts, such as inlay, onlay, osteoperiosteal, or intramedullary and cancellous grafts. Inlay and onlay grafts are used most frequently. The bone graft has been considered a form of rigid internal fixation, and it also assumes a physiological role, giving the host bone additional osteogenic material and providing further stimulus for osteogenesis.

For a time, a great deal of importance was ascribed to the periosteum as a source of osteogenic material in bone-grafting operations. Pollock and Henderson, in 1940, demon-

TABLE VIII

MASSIVE ONLAY GRAFT SECURED WITH CHROMIC CATGUT

Bones	Number of Cases	Results	
		Good	Poor
Humerus	3	0	3
Tibia	1	0	1
Radius and ulna	1	0	1
Ulna	3	1	2
Totals	8	1	7 (87.5%)

strated experimentally that the periosteum contributed little in the formation of new bone. In dogs, where the periosteum was removed from the graft, a fibrous periosteum developed in the surrounding connective tissue. This fibrous periosteum, in four out of five dogs, showed evidence of a more cellular development and more formation of young bone than occurred in those in which the periosteum had not been removed. Clay Ray Murray states that: "New bone will form in the normal adult human tissues only in undifferentiated connective tissue in the presence of available and sufficiently locally concentrated calcium and, possibly still more important, phosphate, and in a medium having a pH not too far to the acid side. . . . In bone grafting, our problem, then, resolves itself into placing a readily vascularizable calcium structure in a freshly prepared rich capillary bed, . . . eliminating movement between the graft and the bone host, and maintaining optimum efficiency in the local minute circulation during the period of incorporation of the graft with the host." Using these principles in the surgical procedures, Murray advises:

1. Preoperatively: To improve the condition of atrophic bones and poor muscles; to treat scars or other skin conditions; and to eliminate infection.
2. Operatively: To have tissues of good viability *in situ*.
3. Postoperatively: Perfect immobilization.

In this paper, it is not intended to go deeply into the surgical technique, since all the details have already been described in the extensive literature on the subject. It is sufficient to mention that practically the only two methods used today are the inlay type of bone graft, used especially by Albee and his followers, and the massive onlay grafts. The inlay, or countersunk, graft came into general use about 1912, and for many years was the method of choice. In 1927 and 1928, Campbell in Memphis, Tennessee, and Henderson in Rochester, Minnesota, developed the method of massive onlay grafts. In 1936, Henderson said: "Experience has taught us that the larger the graft, the better the chances of success". He called his graft the "massive graft", whereas Campbell called it an "onlay graft". It might be more appropriately designated a "massive autogenous onlay graft".

Some changes have been made in the original technique, the principal one being the combined use of massive onlay grafts and vitallium screws to accomplish good internal

TABLE IX

INLAY BONE GRAFT FIXED WITH CHROMIC CATGUT

Bones	Number of Cases	Results	
		Good	Poor
Femur	3	3	0
Tibia	10	5	5
Radius and ulna	6	3	3
Radius	1	0	1
Humerus	1	1	0
Totals	21	12 (57.1%)	9 (42.9%)

TABLE X

INTERVAL BETWEEN INJURY AND TREATMENT

Time Interval	Number of Cases
Less than 6 months	8
6 months to 1 year	38
1 to 2 years	17
2 to 3 years	10
More than 3 years	11
Total	84

fixation. The reports of this technique by Campbell and Boyd in the United States and by Meekison in Canada are sufficiently clear and detailed so that further description is not necessary.

Methods and Techniques

On the 135 cases of non-union in this study, 106 surgical procedures were performed. Tables VI, VII, VIII, and IX show the results of onlay grafts with various modifications. Good results are those with solid union and poor results are those with persistent non-union. In one case of non-union of the radius, a massive onlay graft was used with a plate and vitallium screws. Solid union occurred, but infection was present. In five cases of non-union of the tibia, the use of massive onlay grafts with vitallium screws was combined with inlay grafts. The results were good in three cases; non-union persisted in the other two cases.

These figures indicate that much better results were obtained with grafts secured with vitallium screws, and imply that the important factor is adequate immobilization of the graft.

For more than twenty years, Campbell has been using bone pegs to secure bone grafts. Lately the results with vitallium screws, used to fix the grafts, have been so gratifying that the use of bone pegs has been practically abandoned. The vitallium screws provide better mechanical fixation and simplification of technique. Campbell and Boyd report 93.2 per cent. of good results and 6.8 per cent. of unsuccessful ones. In Table VII are noted the results obtained with massive dual onlay bone grafts. These surpass the results obtained by other methods. The results are more significant, since the success with this procedure was obtained in cases where other surgical bone-grafting procedures had failed. The results with one or two massive onlay bone grafts (88.2 per cent. and 87.5 per cent. of good results) give a clear idea of the value of the method, when compared with the 87.5 per cent. of poor results obtained when the graft was fixed with chromic catgut.

The inlay method of Albee is characterized by precision and accuracy in fitting the graft into a prepared bed. The results in this series with inlay bone grafts are shown in Table IX. In seven fractures of the tibia, sliding inlay grafts were fixed with chromic catgut. The results were good in five cases and poor in two. In one case, an inlay bone graft fixed with bone pegs also resulted in failure. Of two cases of pseudarthrosis of the

TABLE XI

POSTOPERATIVE INFECTIONS IN 135 CASES

Bones	Number of Cases	Previous Infection	No Previous Infection	Previous Open Reduction	Previous Compound Fracture
Tibia	5	1	4	3	2
Humerus	1	1	0	1	0
Radius and ulna	3	1	2	2	2
Totals	9 (6.6%)	3 (2.2%)	6 (4.4%)	6 (4.4%)	4 (3.0%)

TABLE XII

FOLLOW-UP AFTER OPERATION

Time	Number of Cases
6 months to 1 year	18
1 to 2 years	29
2 to 3 years	12
More than 3 years	25
Total	84

tibia treated with inlay bone grafts fixed with vitallium screws, one resulted in good union and one in failure. Another case of non-union of the tibia was treated with an inlay sliding bone graft and held with a vitallium screw; this resulted in solid union. Finally, three cases of non-union of the ulna, treated with inlay and onlay bone grafts fixed with chromic catgut, resulted in two failures and one case of solid union.

A consideration of the various techniques shows that the results with massive onlay bone grafts fixed with vitallium screws are obviously more favorable. Another significant point is that, when dealing with an easily exposed bone, like the tibia, the inlay procedure is relatively simple. However, when grafting the small bones or the deeply placed femur, it is a much more difficult problem.

During surgery, it is the policy in this Department to resect the sclerosed bone ends until a good bleeding end is obtained and then, by drilling, to open up the marrow cavity. Occasionally it is necessary to resect a few inches of the affected bone, which results in a shorter limb. It is therefore important to inform the patient, before operation, that his extremity may be shorter than it was before.

The interval between injury and treatment varied from two months to more than three years (Table X); one patient had a pseudarthrosis for more than ten years. Relatively few patients are operated upon within six months of the time of injury. This arbitrary time limit gives the bones the maximum chance for healing before surgical intervention is attempted.

In cases where suppuration was present, no bone-grafting was tried until there had been complete cessation of infection for at least six months. In only one case was grafting attempted within six months, but union was obtained. In nine cases suppurative processes were present after surgery (Table XI), but healing was finally obtained in five of them. The incidence of postoperative infection was 6.6 per cent. Henderson gives an incidence of postoperative infection in similar cases of 18 per cent.[9]

In this Clinic, local sulfonamide therapy is seldom used; in five cases of non-union where sulfonamides were used locally after bone-grafting, there were three cases of successful union but two cases of failure. Emphasis is placed upon careful preoperative preparation of the patient, and asepsis during surgery.

TABLE XIII

RELATION OF AGE TO HEALING IN BONE-GRAFTING

Ages (*Years*)	Surgery (*No. of Cases*)	Results	
		Good	Poor
5 to 10	3	3	0
11 to 20	12	11	1
21 to 30	20	16	4
31 to 40	18	12	6
41 to 50	15	11	4
51 to 60	7	6	1
61 to 70	12	8	4
Totals	87	67	20

TABLE XIV

FAILURES AFTER BONE-GRAFTING

Case No.	Types of Bone Grafts								Drilling	Infection	Screw Only	Fracture of Graft	Sequestrectomy	Results	
	Inlay and Screws	Sliding	Onlay and Screws	Inlay and Bone Pegs	Inlay and Onlay	Dual Onlay	Inlay and Chromic Catgut	Onlay and Chromic Catgut						Good	Poor
1		1	2						1					x	
2			1								2			x	
3				1	2					2					x
4						2	1							x	
5		1								1			2		x
6		1													x
7					1							1			x
8	1					2								x	
9			1			2								x	
10								1							x
11							1								x
12								1 and 2		2					x
13			2				1			2			3		x
14					1 and 2										x
15								1							x
16			1									1			x
17						2		1						x	
18			1			2								x	
19							1								x
20					1	2									x
21						3		1 and 2		3			4		x
Totals														7	14

1 = Original operation or its complication.
2, 3, and 4 = Subsequent operations.

The follow-up of the patients in this series after operation is shown in Table XII.

The age factor in relation to successful results indicated that healing in bone-grafting operations was obtained most frequently between the ages of five and twenty years, in which consolidation failed in only one out of fifteen cases (Table XIII).

In Table XIV are summarized all the failures following bone-grafting; the type of operation, and also the number of operations to which the patient had previously been subjected, are specified. After failure of the first operation, subsequent bone-grafting gave a total of seven cases with good results (33 per cent.) and fourteen cases of persistent failure (67 per cent.).

CONCLUSIONS

1. Non-union is an avoidable complication in the treatment of fractures.
2. Greater adherence to sound principles in the treatment of fractures will lessen the tendency for pseudarthrosis.
3. In treating this complication by bone-grafting, the principles of sound fracture treatment should be employed.
4. The most efficient treatment for non-union of the shafts of the long bones is by massive autogenous onlay bone grafts or massive dual onlay grafts fixed with vitallium screws.

NOTE: Appreciation is extended to Arthur Steindler, M.D., for his kindness and advice in the preparation of this paper.

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FRACTURE-DISLOCATION OF THE THORACOLUMBAR SPINE

With Special Reference to Reduction by Open and Closed Operations*

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This paper is based upon an analysis of forty-three patients, treated in the Orthopaedic Department of the Royal Victoria Infirmary, Newcastle-upon-Tyne, and in allied hospitals in the district, during a period of approximately six years.

In the vast coal-mining district of Northumberland and Durham, 137,000 men are employed in the industry. The nature of their work is hazardous, and the large majority of the patients with industrial injuries admitted to the hospitals are pitmen. Injuries to the spine occur relatively frequently, and the pitman and his family have come to dread the term "fractured spine", bringing with it, as it does, the vision of permanent paralysis and of a life which is truly worse than death. Of course, not all fractured spines are fracture-dislocations, nor are the latter invariably associated with complete paraplegia. The psychological association remains, however; and this may, in part, account for the high rate of incapacity among miners who have sustained compression fractures, fractures of transverse processes, and other less severe injuries.

The seams in these coal fields are low,—two feet is considered an average; the lowest is eighteen inches. The pitman works, crouching in a confined space; the roof constantly shifts or settles; and "falls of stone" or of "roof" are relatively frequent. If crushed by such a fall while lying, any variety of injury may occur. If sitting in a crouched position, the shoulders are forced forward and downward, causing excessive flexion of the trunk and spine, and resulting in anything from a compression fracture of a vertebral body to a complete fracture-dislocation with paraplegia. In hyperflexion of the spine, the relatively fragile vertebral body is compressed by the body above it, so that a hinge action occurs at the intervertebral articulations. If the force is continued, the interspinous and intervertebral ligaments, and possibly the disc, are ruptured; and dislocation of the intervertebral articulations occurs, so that the upper vertebral body moves forward on the lower. It is then that pressure on, or section of, the spinal cord may occur. The lower articular processes of the vertebra above may be transposed to the front of the upper processes of the vertebra below, so that they become locked in luxation and the dislocation cannot be reduced spontaneously. One or more of these processes may be fractured.

INDICATIONS FOR OPERATION

It has been thought that the paraplegia which complicates fracture-dislocation might be due to:

1. Section of the cord, in which case it is incurable;
2. Pressure on, or stretching of, the cord, without section;
3. Hematomyelia or oedema.

It seems doubtful whether so-called spinal shock ever occurs.

If the paraplegia is due to pressure, hemorrhage, or oedema, and therefore remediable, it is reasonable to assume that recovery might be expedited by the reduction of the displacement and the relief of the pressure. The question arises of how one can estimate the cause or degree of trauma to the cord. Unless the paraplegia is partial, it is impossible to tell on neurological grounds alone whether or not there has been a complete severance of the cord; and, as will be shown later, gross bone displacement, if in injuries of the lower lumbar spine, does not necessarily indicate gross cord damage. A further indication for

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reduction, even in cases of fracture-dislocation without paraplegia, would appear to be the complete correction of the skeletal lesion, so that a maximum recovery of function is achieved. In a large proportion of the patients in the present series, therefore, open or closed reduction has been attempted; and a critical analysis of the results is presented. The series is certainly not great enough to justify any definite conclusions on statistical grounds; but certain general impressions are gained, and a few of the cases are worthy of

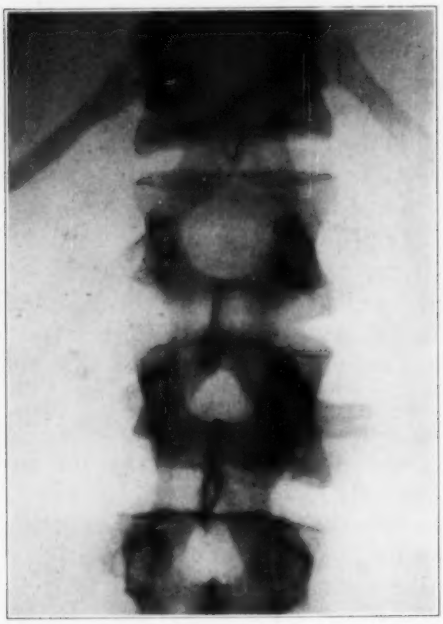

FIG. 1-A

FIG. 1-B

Case 1, G. B. This patient had a fracture-dislocation between the twelfth thoracic and the first lumbar vertebrae, with interlocking of the articular facets.
Figs. 1-A and 1-B: Before reduction.

FIG. 1-C

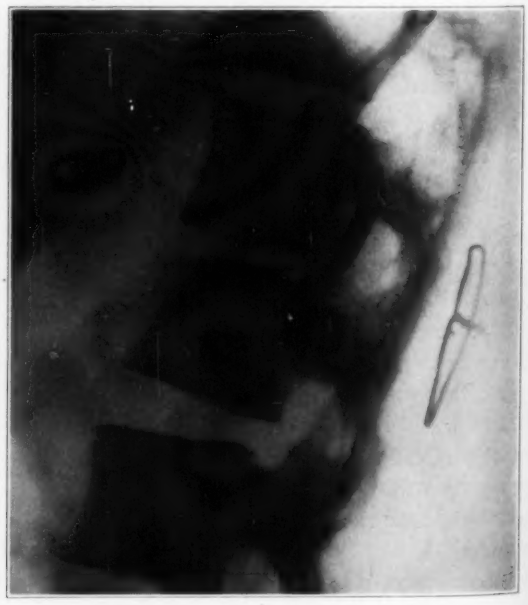

FIG. 1-D

Immediately after reduction.

FIG. 1-E

FIG. 1-F

Present condition, six years later, shows slight redisplacement, but sound bony fusion laterally; the wire has broken.

individual mention. The patients have been treated by several surgeons in the Department, which will account for the variations in the method used.

METHOD OF REDUCTION

Closed reduction was attempted by the author in three patients with complete paraplegia, at a time when it was felt that the outlook in these cases was almost hopeless, and that it was unfair to increase the burden of the patient and of the nursing staff by open operation. The following method was employed:

The patient was laid prone on the operating table, with a pillow under his lower chest, which was placed over the rest employed to hyperextend the spine in abdominal operations. Local infiltration anaesthesia was used. With a moderate degree of traction on the arms and legs, the spine was flexed by the elevation of the rest; at the same time, pressure was exerted over the spine, below the dislocation. Reduction was accompanied in each case by the usual satisfactory sensation which one associates with the reduction of any dislocation, and the spinous processes were felt to assume their normal relationships.

One must admit, on theoretical grounds at least, that *open* reduction of the interlocking articular facets, under the careful control of one's eye, is the method of choice. It is a relatively simple procedure, which should be done under local infiltration anaesthesia. The muscles can always be stripped off the damaged vertebrae with the greatest of ease, having been separated by the trauma and subsequent hemorrhage. The operation should be preceded by very careful roentgenography to ascertain the exact extent of the damage, particularly that to the posterior structures. The interlocking articular processes are readily identified, the cartilage of the lower facets gleaming white when the wound is swabbed. If the dislocation is in the lower thoracic region, the processes tend to be perched upon one another, tip to tip. Little forward displacement occurs at this site, because the spinous process of the upper vertebra is very oblique, and, if unfractured, will not permit displacement. It is not unusual to find the articular facets already unlocked and commencing to mate, requiring only extension of the spine to complete the process. In the lumbar region, however, true interlocking occurs. It has been recommended

that partial removal of the upper facets of the lower vertebra be performed, on one or both sides, to facilitate reduction without undue manipulation or roughness. The writer only rarely found this necessary. Gentle flexion of the spine to unlock the facets, with backward traction on the spinous process of the upper vertebra, has been enough to secure unlocking; and accurate reposition is then effected by hyperextension. On one occasion the spinous processes of the two affected vertebrae were wired together to prevent redisplacement. This was in a patient with no paraplegia. The wire has not affected the functional recovery, which is complete. The man served in the Army overseas throughout the War, and was classified as A.1 on entry, within eight months of his accident (Figs. 1-A to 1-F, inclusive).

On both the open and closed operations, reduction is maintained by the application of a plaster cast. It there is no fracture of the posterior structures, a complete jacket is applied. If there is any doubt about the integrity of the posterior column, a plaster boat is felt to be safer, following reduction.

SITE OF LESION

Has the degree of cord injury any relationship to the site of the lesion, or to the degree of trauma to the spine? It will be seen from Table I that the junction of the fixed and movable parts—that is, the thoracolumbar region—is the most important site of injury. Of the major group of thirty-six cases in which the fracture-dislocation occurred near the thoracolumbar junction, twenty-five patients had complete paraplegia; three had partial paraplegia; and eight had no paraplegia. Of the small group of five cases in which the lesion occurred at or below the level of the second and third lumbar vertebrae, however, two with very gross displacement were complicated by complete paraplegia; one, with interlocking, by partial paraplegia; and two patients, one of whom showed the grossest displacement between the fourth and fifth lumbar vertebrae, had no paraplegia (Fig. 2).

FIG. 2

Case 2, L. S. This is a case of fracture-dislocation between the fourth and fifth lumbar vertebrae, with gross displacement. There was no paraplegia.

This relatively low incidence of nerve damage in the group whose lower spine was involved must be due to (a) the large size of the neural canal and (b) the greater strength of the cauda equina as compared with the spinal medulla. Gross displacement at the thoracolumbar junction, however, must indicate severe cord injury, although the converse—that is, slight displacement with simple interlocking—is no indication whatever of the degree of cord damage.

The case labeled "None, becoming complete" (Table II) is of especial importance. This case was diagnosed upon admission to

TABLE I

SITE OF FRACTURE-DISLOCATION OF THORACOLUMBAR SPINE IN FORTY-THREE CASES

Vertebrae Involved	Number of Cases
Eighth and ninth thoracic vertebrae	1
Ninth and tenth thoracic vertebrae	1
Eleventh and twelfth thoracic vertebrae	13
Twelfth thoracic and first lumbar vertebrae	17
First and second lumbar vertebrae	6
Second and third lumbar vertebrae	2
Third and fourth lumbar vertebrae	2
Fourth and fifth lumbar vertebrae	1

the hospital as a compression fracture of the second lumbar vertebra; the following day, under a general anaesthetic, the patient was treated by hyperextension and plaster-of-Paris. When he recovered from the anaesthetic, he had a complete paraplegia. Within four hours of this treatment, an open reduction of a fracture-dislocation between the first and second lumbar vertebrae was performed; and, apart from some weakness of the dorsiflexors of his left foot, the patient has made a complete recovery from the paraplegia. This recovery commenced within four days of the reduction. This mistake was due to bad roentgenography and also to faulty interpretation of the plates (Figs. 3-A to 3-H, inclusive).

RESULTS

Closed Reduction

Of the five cases with complete paraplegia treated by the closed method (Table III), one patient made a complete recovery, is back at light work, and is a wrestler of some repute. Another made a partial recovery, has good hip and knee control, but a bilateral paralytic talipes calcaneus; after surgical treatment for this, he will be able to walk again (Figs. 4-A to 4-E, inclusive). A third patient showed some early promise, and within four months had partial sensory and motor recovery in both lower limbs; he was able to take a few steps before it was necessary to evacuate him because of war conditions. He died very recently; unfortunately an examination was not possible before his death, but he apparently made no further recovery after leaving the hospital. No details concerning the other two patients can be given, as they were not treated by the writer. There is no record that reduction was successful, nor did any recovery occur.

One patient with partial paraplegia was treated by manipulative reduction, and made an almost complete recovery. Apart from bilateral drop-foot, he has good voluntary control of all muscles. Roentgenograms show that reduction is good, and spontaneous fusion of the two affected vertebrae has occurred. He is doing light work.

The four patients with no paraplegia were treated by hyperextension and plaster, presumably under the impression that they had compression fractures. No disasters resulted, as might have been expected. Indeed, in one case reduction was accomplished by this method, but the spine has subsequently become displaced again. All four patients are sufficiently fit to do light work.

TABLE II

DEGREE OF PARAPLEGIA COMPLICATING THORACOLUMBAR FRACTURE-DISLOCATIONS

Paraplegia	Number of Cases
None	8
Partial	3
Complete	27
None, becoming complete	1
Partial, becoming complete	2
Partial, disappearing on or before admission (based on statement of patient)	2

Open Reduction

The results in those patients treated by open operation have been very disappointing. Of eighteen patients with complete paraplegia, only one has made an almost complete recovery from the nerve injury. He was the patient mentioned previously, in whom a complete paraplegia developed after treatment of an uncomplicated fracture-dislocation

FIG. 3-A

FIG. 3-B

Case 4, B. C. This patient had a fracture-dislocation between the first and second lumbar vertebrae, with interlocking of the articular facets and lateral shift.
Figs. 3-A and 3-B: Before reduction.

FIG. 3-C

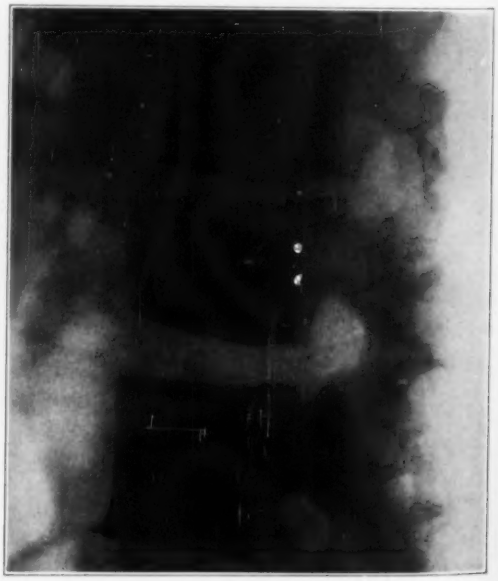

FIG. 3-D

Immediately after reduction.

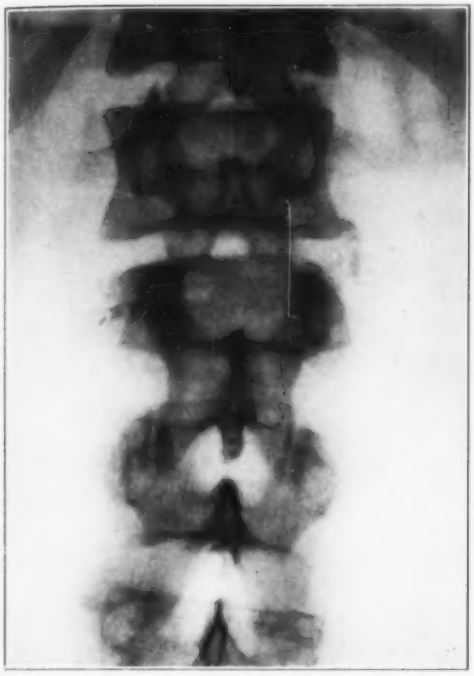

FIG. 3-E

FIG. 3-F

Six months after reduction.

FIG. 3-G

FIG. 3-H

Present condition, five years after reduction; roentgenograms show redisplacement, but sound lateral and anterior fusion.

by hyperextension under a general anaesthetic. A recent review shows that the dislocation has recurred, without any recurrence of the paraplegia (Figs. 3-G and 3-H). Twelve patients are dead, but six are reported to have shown very slight signs of recovery. Of the other five still living, not one has shown any signs of recovery.

Of the two patients with partial paraplegia who were treated by open reduction, one has made a good degree of recovery, and, in spite of partial incontinence of urine, is doing light work. The other has made slight recovery, and is walking with a caliper brace.

Of the five patients with no paraplegia who were treated by open reduction, two have

FIG. 4-A

FIG. 4-B

FIG. 4-C

Case 5, R. H. This patient had a fracture-dislocation between the twelfth thoracic and first lumbar vertebrae, with interlocking of the articular facets.
Figs. 4-A and 4-B: Before reduction.
Fig. 4-C: Immediately after reduction (only lateral view available).

FIG. 4-D

FIG. 4-E

Present condition, six years after reduction; roentgenograms show complete redisplacement, but sound lateral fusion.

made a complete functional recovery from the skeletal lesion and are back at full work. The others showed good results, but are doing only light work.

No Reduction

Seven patients with complete paraplegia were not treated by reduction, open or closed, but simply by general nursing. Six of them showed no sign of recovery; but the most recent

FIG. 5-A

FIG. 5-B

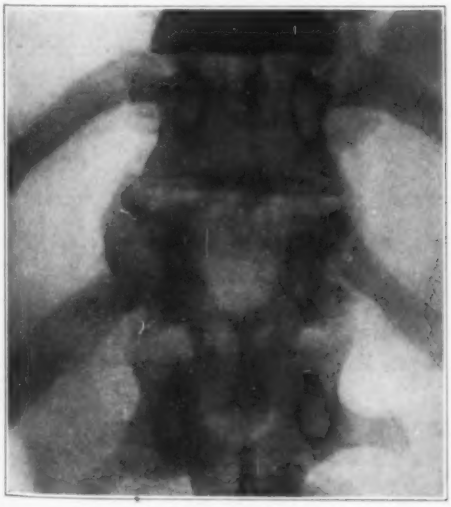

FIG. 5-C

FIG. 5-D

Case 6, E. F. This patient had a fracture-dislocation between the eleventh and twelfth thoracic vertebrae, with interlocking of the articular facets. Figs. 5-A and 5-B: Condition on admission. Figs. 5-C and 5-D: Present condition, ten months later. These films show that no reduction has occurred, yet this patient's paraplegia shows signs that a complete recovery may be expected.

TABLE III

RESULTS

Degree of Paraplegia	No. of Cases	Method of Reduction		
		Closed	Open	No Reduction
Complete	30	5 (1 Hyperextension)	18	7
Partial	3	1	2	0
None	10	4 (4 Hyperextension, 1 Successful)	5	1
Totals	43	10	25	8

one will probably make a complete, or almost complete, recovery. This patient sustained a fracture-dislocation between the eleventh and twelfth thoracic vertebrae, with interlocking of the articular processes and a complete paraplegia. He was very ill on admission, and for this reason it was not thought worth while to attempt reduction. Six weeks after the injury, however, signs of motor recovery in both lower limbs were noted, followed by some control of the sphincters after three months. He was walking within three months of his injury; now, ten months later, he is at a Rehabilitation Center, and it is anticipated that he will make an almost complete functional recovery. This case would seem to indicate that the possibility of recovery in a complete paraplegia does not depend upon the reduction, whether open or closed, but upon the initial degree of injury to the spinal cord. This is still presumably stretched and attenuated, yet recovery is occurring (Figs. 5-A, 5-B, 5-C, and 5-D).

Signs of Recovery

The estimation of signs of neurological recovery in completely paraplegic patients is always a matter of great difficulty in the early stages. Unless approached in a very critical fashion, fallacious signs of recovery may be recorded. Patients will, for instance, move the foot by dragging and pushing a paraplegic limb, if the heel is not supported clear of the mattress. The patient is always anxious to please, and, unless questioned carefully, will admit sensation which is not really felt.

The earliest signs of recovery have been recorded as occurring immediately after reduction in some instances; in others, as late as three months after reduction. Sensation seems to be recovered earlier than muscle power. *The early appearance of recovery seems to be no indication of the eventual prognosis*; in the patients showing almost complete recovery, the first signs were noted as early as four hours after reduction or as late as six weeks.

Although treated by immobilization in plaster for a period of about four to six months, several of those patients in whom complete reduction has been performed have, in the recent survey, been found to have redislocations (Figs. 3-A to 3-F and 4-A to 4-E).

Redisplacement does not necessarily mean a painful back, however. Indeed, this has been followed in all instances by spontaneous bony fusion between the two affected vertebrae, and the functional result has been excellent. Furthermore, there has been no evidence that redislocation has been followed by further signs of nerve damage.

Complications

In two cases, a most interesting complication was observed. This consisted of an extensive myositis ossificans in the pelvifemoral muscles around both hip joints (Figs. 6 and 7). This was noted in both instances about six months after the accident. The masseuse treating both patients noticed an increasing resistance to passive movements at the

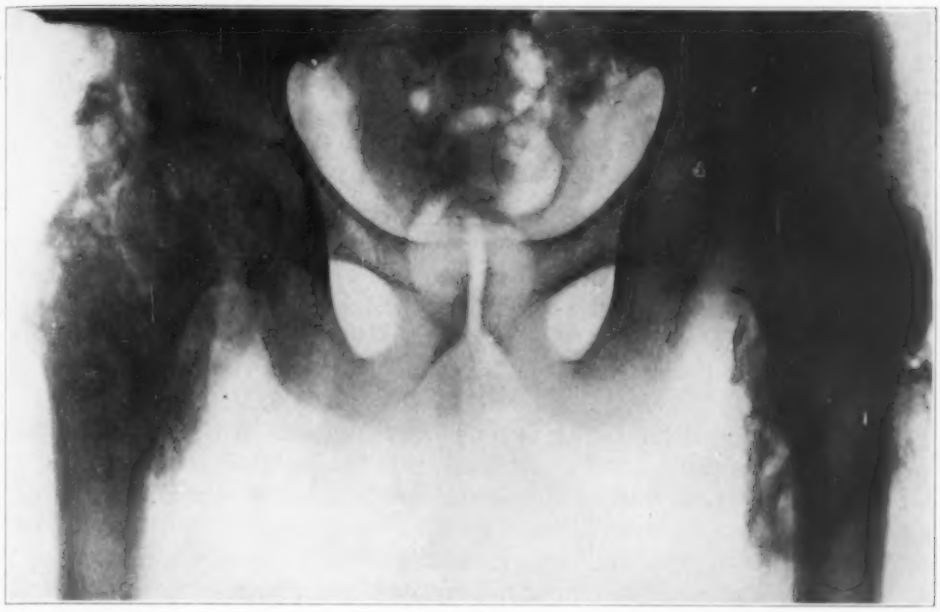

FIG. 6

Case 7, J. W. This is a case of fracture-dislocation between the eleventh and twelfth thoracic vertebrae, with complete paraplegia; the patient did not recover. This film shows extensive myositis ossificans around both hip joints.

FIG. 7

Case 8, R. S. This is a case of fracture-dislocation between the first and second lumbar vertebrae, with complete paraplegia; the patient did not recover. The film shows extensive myositis ossificans around both hip joints.

hips, commencing about the fourth month. In one case, there was no evidence of a similar condition elsewhere in the body; in the other, there was an abnormal ossification in the region of the knee joint, and a large stone subsequently developed in the patient's kidney. In both instances, the urea excretion was poor; but no abnormality in the blood calcium, phosphorus, or phosphatase was noted. It has been suggested that this myositis ossificans may be due to one or both of the following factors:

1. The impaired urinary excretion of nitrogenous bodies, causing a mobilization of calcium from the bones of the paralytic limbs. The normal blood chemistry is against this, although it must be added that experiments to determine calcium and phosphorus balance were impossible because of wartime conditions.

2. A Charcot-like reaction around a denervated joint, subjected to the repeated minor traumata of passive stretching by the masseuse.

CONCLUSIONS

1. The site of the lesion determines, to some extent, the possibility and the degree of paraplegia. There is a higher relative incidence of paraplegia at the thoracolumbar junction than in the lower lumbar region. This presumably is due to (a) the larger size of the neural canal, and (b) the toughness of the cauda equina, as compared with the spinal medulla.

2. The extent of skeletal damage is no indication of the degree of cord trauma. For instance, in the thoracolumbar region, a simple interlocking of the articular facets, with a minimum anteroposterior shift, is often associated with complete paraplegia; yet at this site complete displacement must indicate complete transection of the cord. In the lower lumbar region, however, gross displacement may be present without paraplegia.

3. The method of reduction seems to play little or no part in the possibility of recovery. In the small series under discussion, closed reduction seemed to give better results than open, but it would appear to be doubtful whether reduction matters at all.

4. The prognosis, if reasonable care is taken not to inflict further damage on the cord by careless handling, is surely decided at the time of the initial trauma.

5. The idea is disproved that stretching or attenuation of the cord may be an important cause in preventing recovery from paraplegia in cases of fracture-dislocation with interlocking facets.

6. The early appearance of the first signs of recovery of sensation or of motor power is no indication of the eventual prognosis.

7. Hyperextension, particularly with anaesthesia, which produces complete muscle relaxation, is to be condemned. In the present series, however, it has been tried in error on several occasions *without anaesthesia*, without disaster; and in one case it resulted in reducing the fracture-dislocation.

8. In the series examined, the dislocation, if reduced, has very often recurred, but without further signs of cord injury. Furthermore, this redisplacement is commonly associated with spontaneous anterior or lateral fusion of the vertebral bodies, giving rise to an excellent skeletal function.

One has not the courage, on the basis of such limited experience, to advise against reduction in these cases; but an optimistic attitude must be adopted, whether reduction is performed or not. Careful nursing and physiotherapy are necessary from the beginning. These patients must no longer be discharged from busy surgical wards on the grounds that the outlook is hopeless.

OSTEOTOMY-OSTEOCLASIS

A Method for Correcting Long-Bone Deformities

BY JOHN ROYAL MOORE, M.D., PHILADELPHIA, PENNSYLVANIA

Numerous methods for the correction of deformities of the long bones are available in recent and old textbooks and in the literature. In 1878 Macewen described what is now known as the cuneiform osteotomy, employing it at that time for the correction of knock-knee deformities. Numerous other types of osteotomies including linear, circular, block, pivot, Z, telescoping, *et cetera* have been described. One of the principal difficulties in the correction of long-bone deformities has been the problem of prevention of displacement of the fragments. This factor, more than any other, has been largely responsible for the innumerable types of osteotomies, only a few of which have been mentioned in the preceding sentence. In recent years, internal fixation has been used to supplement the various types of osteotomy and has gone far toward eliminating the difficulty of slipping. However, the necessity of larger incisions, the usual hazards associated with more difficult surgery, the increased danger of infection, and the risk of necrosis (electrolytic, thermal, and ischaemic) have detracted considerably from the mechanical advantages.

The osteotomy-osteoclasis method consists simply in cutting the long bone almost entirely in two, near or at the site of the maximum deformity, and then completing the osteotomy by manual osteoclasis at a later period (twenty-one days in growing bone and twenty-eight days in adult bone). The osteotomy-osteoclasis routine was first employed upon a severely malunited subtrochanteric femoral fracture, at the Shriners' Hospital for Crippled Children, Philadelphia, Pennsylvania, in 1934. A careful review of the literature has supplied no reference to this osteotomy-osteoclasis routine, but it is highly probable that it has already been described.

Osteotomy and osteoclasis are well-known procedures, usually done as separate operations and occasionally combined at one operation. In July 1939, Ferguson, Thompson, and King reported a two-stage osteotomy.

A total of 225 operations of this type have been performed to date. The long-bone deformities consisted of malunited fractures, ankylosed joints with severe deformity of one type or another, knock-knee, bow legs, coxa vara, humerus varus, cubitus varus, *et cetera*.

TECHNIQUE

A characteristic of the method is its simplicity. The site of the maximum deformity is approached through the most direct route. A linear incision, six to eight centimeters long, is made over the site of the deformity. Routine surgical technique is employed through subcutaneous tissue, fascia, and muscle, and down as far as the periosteum. The periosteum is then split carefully and preserved with the utmost care. A wedge of bone is removed, either at the site of the maximum deformity or just above or just below it (the osteotomy

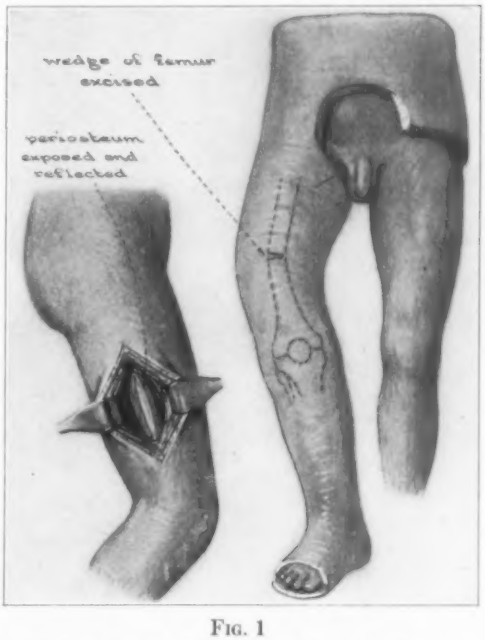

FIG. 1

Drawing illustrating incision, incomplete osteotomy, and immobilizing cast.

should be made through healthy bone and, therefore, when the bone at the site of the deformity is abnormal, it is necessary to go just above or just below the site of the maximum deformity). The base of the wedge is determined from the preliminary roentgenogram and, as a matter of routine, it is always slightly larger than necessary in order to ensure an easy correction. The wedge includes the cortex on the three sides of the bone. The cortex remaining on the one side is drilled once or twice with a three-sixteenths-inch drill. Very gentle pressure is then applied to the bone in question to make sure that slight bending occurs at the site of the osteotomy. The wedge of bone is then cut up into small pieces, and replaced in the defect. The periosteum is carefully closed. This is a simple matter in growing bone, but in adult bone it requires more care. Often the periosteum is only a filament-like layer attached to the adjacent muscle, but it will still serve as a limiting membrane and possibly as an osteogenic layer if properly protected. The remaining soft parts are closed in a routine manner; the usual

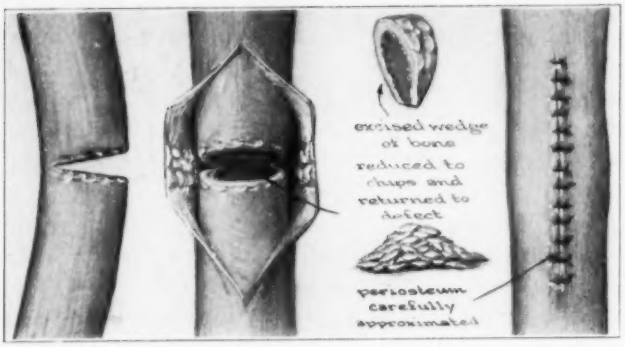

FIG. 2

Sketch showing wedge, chips, and careful closure of the periosteum.

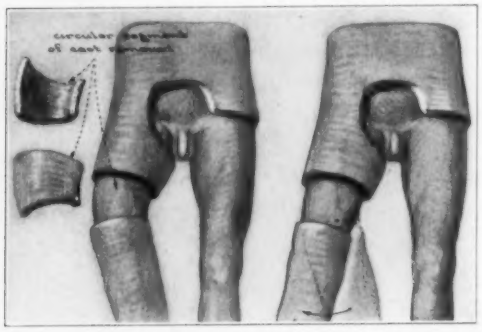

FIG. 3

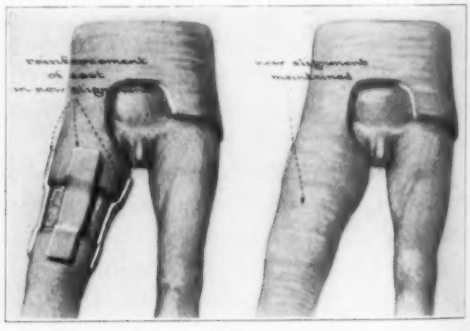

FIG. 4

Fig. 3: Sketch illustrating removal of section of cast in preparation for osteoclasis.
Fig. 4: Cast following osteoclasis. Note circular section being repaired.

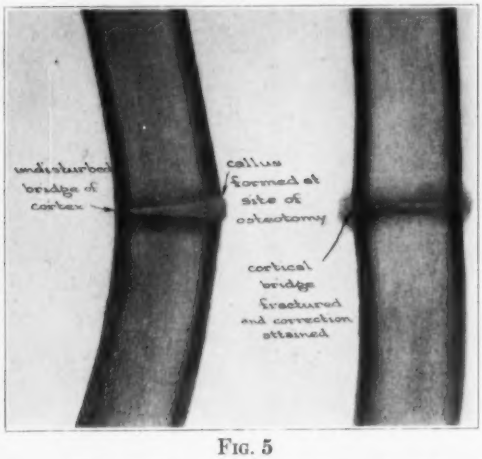

FIG. 5

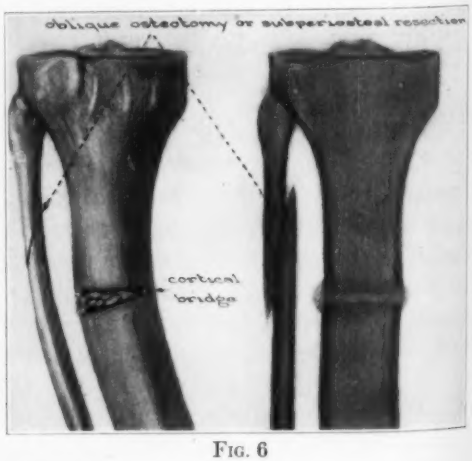

FIG. 6

Fig. 5: Sketch showing ring callus following osteoclasis.
Fig. 6: Showing special technique employed in deformities of the upper end of the tibia.

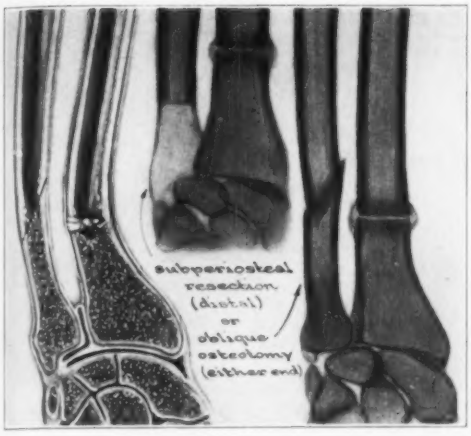

FIG. 7

Fig. 7: Sketch illustrating technique employed in deformities of the lower end of the tibia.

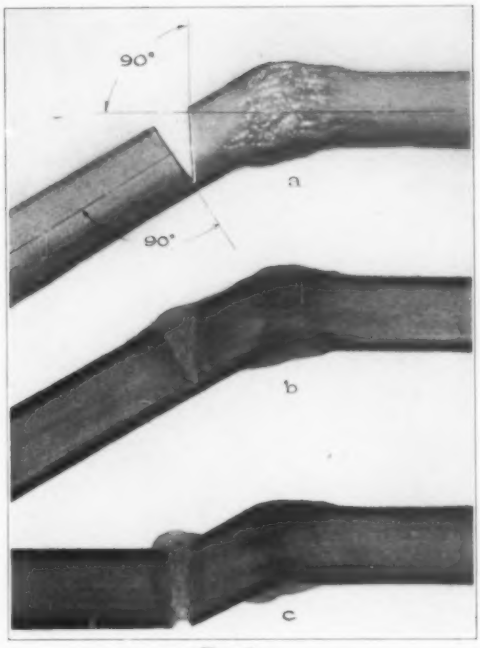

FIG. 8

Fig. 8: Incomplete wedge osteotomy being employed just below fracture site. Bone at fracture site is sclerotic and is not considered proper for osteotomy.

dressing is applied; and a plaster-of-Paris splint or cast is applied, including the joint below and the joint above the osteotomy site. The anaesthesia period is rarely longer than ten or fifteen minutes and any of the standard anaesthetics may be employed.

At the end of twenty-one days in an individual with growing bone and twenty-eight days in the adult, the patient is again returned to the hospital; and a cylindrical section of the cast is removed at the site of the incomplete osteotomy. The true axis of the wedge is determined by preliminary roentgenograms and, if necessary, it is checked by fluoroscope. The patient is given a light anaesthetic, and the osteoclasis is performed. It is extremely important that the axis of the wedge be determined, so that the gentle force takes place along the proper axis. The intact bone is easily broken. The correction of the deformity is then readily accomplished, and with a minimal amount of displacement of the fragments. The defect in the plaster is then filled in with slabs and circular plaster. The long bone of the child is held for an additional six weeks and that of the adult for eight to twelve weeks, depending upon the level of the fracture (fracture in midshaft in an adult for twelve weeks, cancellous bone in an adult for eight weeks).

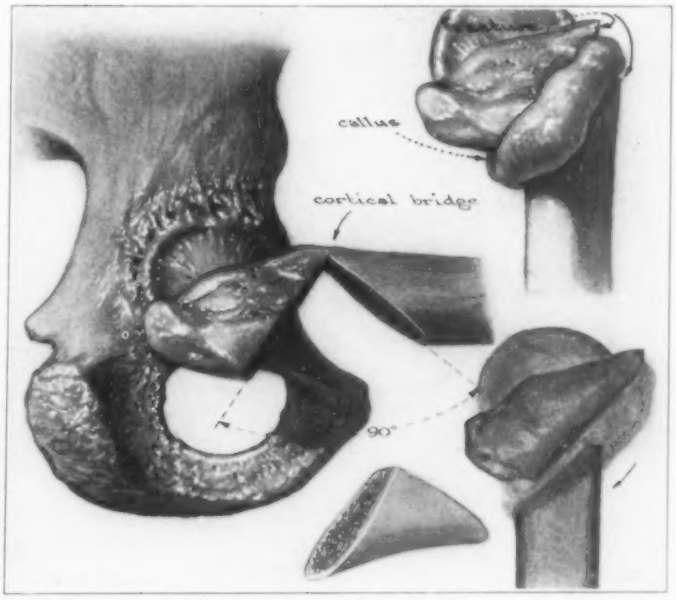

FIG. 9

Sketch illustrating technique employed in severe flexion deformity at the hip. Note location of wedge.

FIG. 10-B

FIG. 10-A

Fig. 10-A: E. M., aged thirteen years. Anteroposterior and lateral roentgenograms, taken February 8, 1944, showing severe bowing deformity of the left femur, middle third.
Fig. 10-B: February 16, 1944. Anteroposterior and lateral views, showing the incomplete osteotomy.

Fig. 10-A: E. M., aged thirteen years. Anteroposterior and lateral roentgenograms, taken February 8, 1944, showing severe bowing deformity of the left femur, middle third.

Fig. 10-B: February 16, 1944. Anteroposterior and lateral views, showing the incomplete osteotomy.

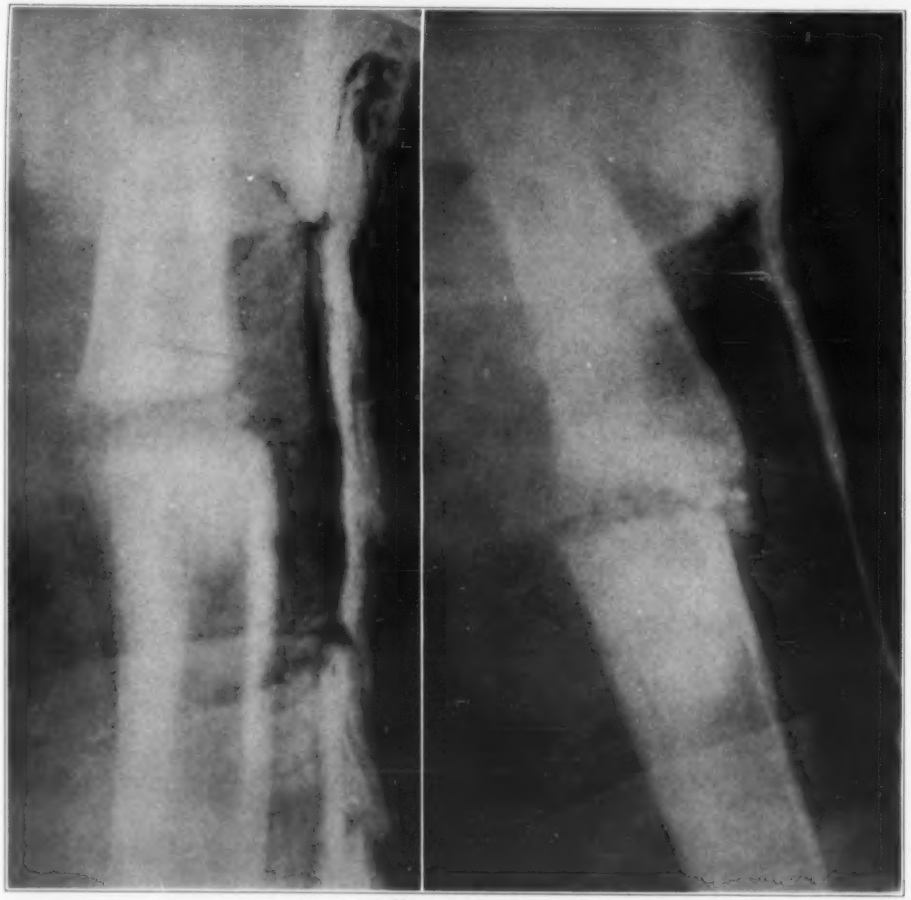

FIG. 10-C

FIG. 10-D

Fig. 10-C: Anteroposterior and lateral roentgenograms, March 14, 1944, showing osteoclasis.

Fig. 10-D: Anteroposterior and lateral views, January 25, 1945, nine months following osteoclasis. Note perfect alignment and absence of displacement.

FIG. 11-A

FIG. 11-B

Fig. 11-A: Mrs. C. S., aged thirty-seven years. Roentgenogram, taken May 22, 1944, showing incomplete osteotomy at the junction of the middle and upper thirds of the shaft of the femur.

Fig. 11-B: June 28, 1945. Anteroposterior and lateral views of the osteotomy site, approximately twelve months following osteoclasis. Note absence of displacement.

RESULTS

Of the 225 cases reported, there has been no evidence of non-union, malunion, or slipping or displacement of fragments at the time of the osteoclasis. There has been no evidence of fat embolism or embolic phenomena of any kind. There has been no circulatory

FIG. 12-A

J. E., aged twenty-one years. Roentgenogram, taken May 10, 1944, showing bony ankylosis of the right hip. Note adduction deformity; severe 80-degree flexion deformity was also present.

FIG. 12-B

Lateral and anteroposterior views of osteotomy site, October 3, 1944, five months later. Note union, excellent alignment, and lack of displacement.

Fig. 11-A: Mrs. C. S., aged thirty-seven years. Roentgenogram, taken May 22, 1944, showing incomplete osteotomy at the junction of the middle and upper thirds of the shaft of the femur.
Fig. 11-B: June 28, 1945. Anteroposterior and lateral views of the osteotomy site, approximately twelve months following osteoclasis. Note absence of displacement.

FIG. 13-A

FIG. 13-B

Fig. 13-A: C. H., thirteen months old. Anteroposterior and lateral roentgenograms taken November 25, 1944, showing congenital anterior and medial bowing deformity at the lower end of the left tibia.

Fig. 13-B: June 13, 1946. Anteroposterior and lateral views of the left tibia and fibula, eighteen months after the osteoclasis. Note the absence of any displacement. Slight lateral bowing still persists.

deformity at the lower end of the left tibia.

Fig. 13-B: June 13, 1946. Anteroposterior and lateral views of the left tibia and fibula, eighteen months after the osteoclasis. Note the absence of any displacement. Slight lateral bowing still persists.

FIG. 14-A

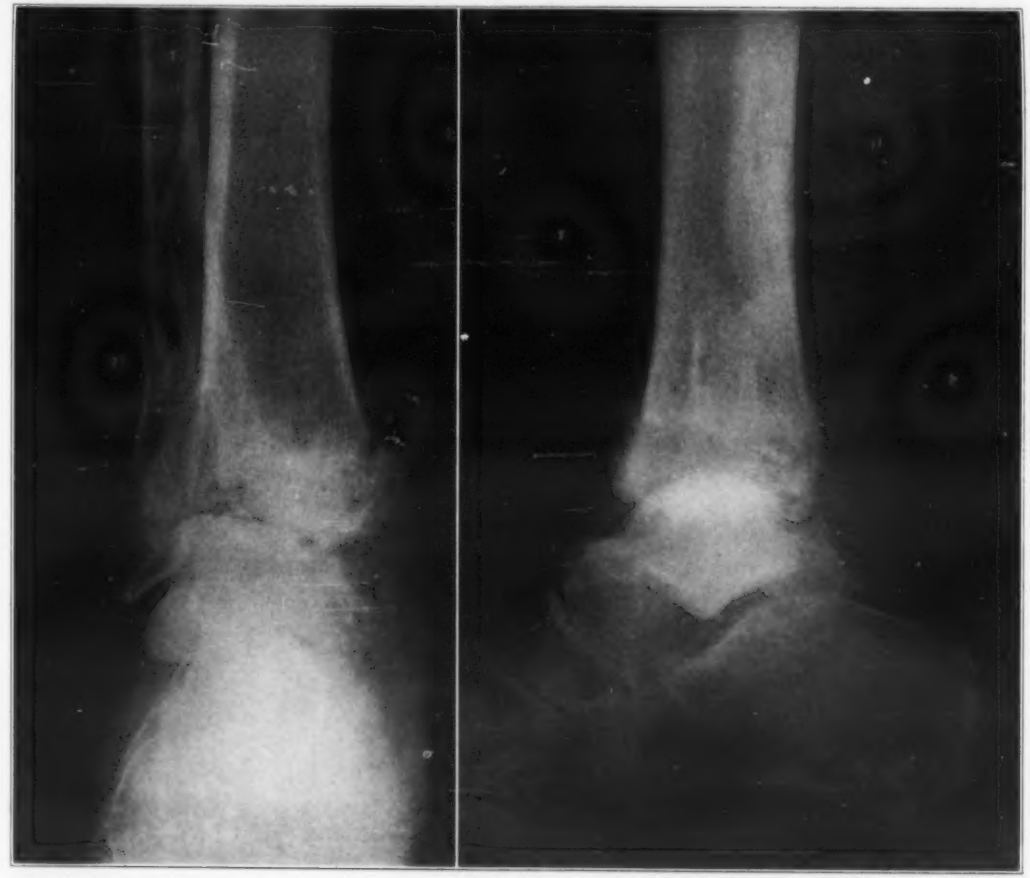

FIG. 14-B

Fig. 14-A: J. H., aged twenty-four. Anteroposterior view of the right ankle joint, taken January 10, 1946, shows bony ankylosis of the talotibial joint and malunited bimalleolar fracture.

Fig. 14-B: May 16, 1946. Anteroposterior and lateral views of the right ankle, five months after osteoclasis.

FIG. 15-A

FIG. 15-B

Fig. 15-A: A. K., aged fifteen years. May 20, 1943. Anteroposterior roentgenogram of the right humerus, upper third, illustrating a severe lateral bowing deformity.

Fig. 15-B: September 22, 1943. Anteroposterior view, four months later. Note the correction of the deformity, lack of displacement, and early evidence of repair.

disturbance,—venous, arterial, or lymphatic. The sketches are illustrative of the method; and the roentgenograms show some of the typical results.

DISCUSSION

Osteotomy-osteoclasis is extremely valuable in the correction of angulations, bowings, and rotary deformities of long bones. It obviously cannot be employed to gain length, other than the apparent lengthening that follows the correction of the angulation of a bowing deformity. The incision employed is small; and the closure is extremely simple, since there is no distortion of soft parts, because the correction of the deformity is done immediately. The approach is simple; and the time required for the wedge osteotomy is only a few minutes. This minimizes the risk to older people.

The author has employed the procedure a number of times in the correction of deformities of people beyond sixty years of age. The fact that there are two periods of anaesthesia is objectionable, but the total anaesthesia time is much below the total employed in the usual osteotomy in the adult. It is not usually necessary to apply the splint or the plaster at the time of operation; this can be added two or three days after the operation if necessary. In no instance has the patient objected to the second anaesthesia. Follow-up roentgenograms are taken immediately after the osteotomy and, in case it is too low or too high, the defect may be allowed to heal and the osteotomy may be repeated at the proper level at a later period. In case there is infection in the neighborhood—such as about the hip or the knee—which does not respond to treatment, hence theoretically postponing correction of a severe deformity indefinitely, it is possible to do the incomplete osteotomy at a slightly lower level with a feeling of safety; and, should infection occur, it is considerably less dangerous, because the osteotomy is incomplete. The osteotomy may serve as a means of testing a dormant infection in an old scar. The delayed osteotomy produces distinctly less shock, and hemorrhages have never been an accompanying problem. In case the deformity involves two bones, as in the forearm or leg, it is necessary to do the incomplete osteotomy on both of the bones and preferably at the same level, and, of course, in the same axis. In deformities of the upper tibia, a resection of the proximal end of the fibula facilitates the osteotomy-osteoclasis of the tibia. The fact that there is no appreciable displacement at the time of the osteotomy should be strongly emphasized. Another important point is the fact that an absolutely perfect alignment can be obtained through a check by fluoroscope (no correction is done under fluoroscope). The fact that

the wound is closed makes it possible to correct the angulation or bowing in both planes, and to correct the rotary deformity in a way similar to that used in any flexible tubing.

CONCLUSIONS

The writer feels that the method for the correction of long-bone deformities described is fundamentally sound; that it is applicable in the correction of any of the long-bone deformities; and that it should be employed to the exclusion of major surgery and extensive internal fixation whenever possible.

NOTE: Even though the first osteotomy-osteoclasis was performed in 1934 when a preliminary report was given by the writer, twelve years were allowed to elapse, in order to accumulate a sufficiently large group of cases for an analysis of the results.

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FRACTURE-DISLOCATION OF THE ANKLE WITH FIXED DISPLACEMENT OF THE FIBULA BEHIND THE TIBIA*

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From St. Luke's Hospital, New York City

In the past eighteen months, five cases of fracture-dislocation of the ankle joint have been seen, in which satisfactory reduction could not be accomplished by closed methods. On open exposure, a situation was found which, to the author's knowledge, has not previously been reported. It is probable that many other such cases occur, and that bad results ensue, due to lack of knowledge of the basic mechanical situation.

The first patient was seen in December 1944. Roentgenograms showed a typical bimalleolar fracture, with marked displacement (Fig. 1-B). On attempted closed reduction,

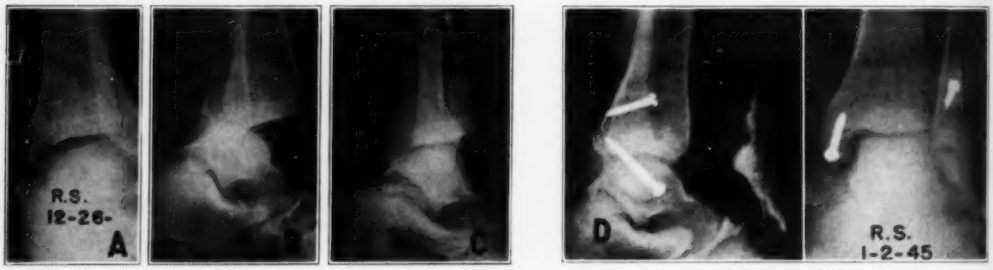

FIG. 1-A FIG. 1-B FIG. 1-C FIG. 1-D

Fig. 1-A: Faulty reduction, secured in fracture-dislocation of the ankle when the upper fibular fragment was caught behind the tibia.
Fig. 1-B: Original roentgenogram of fracture-dislocation of the ankle.
Fig. 1-C: Lateral view, following attempt at closed reduction, shows faulty position of talus beneath tibia.
Fig. 1-D: Postoperative reduction, after fibula had been pried from behind tibia and the fragments had been fastened with screws.

poor reposition of the talus beneath the tibia was secured, and even this could not be maintained (Figs. 1-A and 1-C). An open exploration was done, and even at operation it was impossible to determine at first why reduction could not be secured. Upon further extension of the incision, it was found that the proximal portion of the fibula had been displaced behind the tibia and was caught in back of the posterolateral ridge of the tibia. The fibula was held in this position by the tight stress of the interosseous membrane above. A pry was inserted between the tibia and fibula; and, with considerable force, the fibula was pried out from in back of the tibia and moved into its proper place with a loud snap. Reduction of the fracture-dislocation was then secured easily.

In February 1945, Henry Briggin, M.D., called about a patient with fracture-dislocation of the ankle in whom the fibula alone was involved. He had attempted to do two reductions by aid of the fluoroscope within half an hour of the time of injury, but was unable to replace the talus satisfactorily beneath the tibia. Indeed, when he had obtained the reduction as nearly as possible, the blood supply to the rest of the foot was lacking. A diagnosis was immediately made of posterior fixation of the fibula behind the tibia; and at operation, a few hours later, this situation was found to be present. The fibula was pried out from its displaced location, and the talus could easily be replaced beneath the tibial border (Figs. 2-A, 2-B, and 2-C). In both of these instances, metal fixation of the fractured malleoli was done.

* Read before the Orthopaedic Section of the New York Academy of Medicine, New York City, May 18, 1945; and at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 28, 1946.

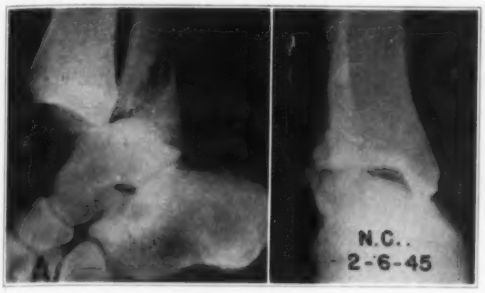

FIG. 2-A

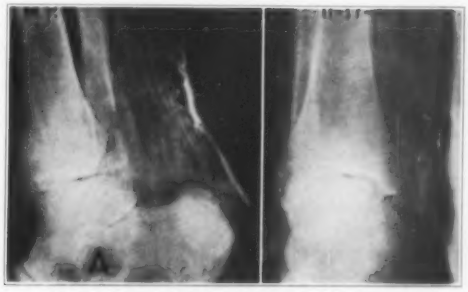

FIG. 3-A

FIG. 2-B

FIG. 3-B

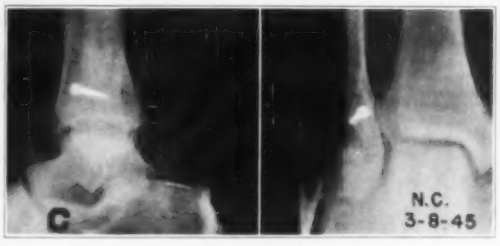

FIG. 2-C

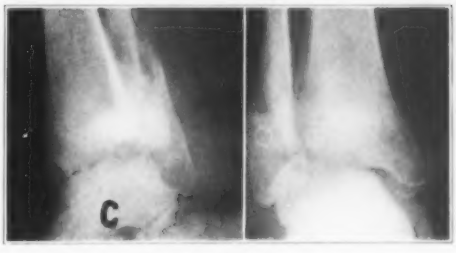

FIG. 3-C

Fig. 2-A: Fracture-dislocation of the ankle before reduction, with fibula caught behind posterolateral ridge on tibia.

Fig. 2-B: Showing the best closed reduction that could be secured. At the time these roentgenograms were taken, the foot circulation was shut off by mechanical pressure of the fibula against the posterior vessels.

Fig. 2-C: Reduction has been obtained by open operation, use of a pry, and fixation of the lateral malleolus with a screw.

Fig. 3-A: Original reduction of fracture-dislocation of the ankle, with fibula caught behind tibia. Roentgenograms show incomplete reposition of talus beneath tibia.

Fig. 3-B: Result of fifth attempt at closed reduction. Faulty reposition of talus under tibia and widened ankle mortise are shown.

Fig. 3-C: Final result in a fracture-dislocation of the ankle, with fibula caught behind tibia. Attempt at closed reduction was unsatisfactory. Solid malunion was present before ankle fusion was done.

The third case was seen at the Mary Fletcher Hospital in Burlington, Vermont. The patient appeared with the typical clinical findings of malunion of a fracture-dislocation of the ankle, which involved the lateral malleolus alone. Roentgenograms showed incomplete reduction of the talus beneath the tibia and the typical findings of posterior displacement of the upper fragment of the fibula behind the tibia, although to the casual observer the fibula did not appear so displaced (Figs. 3-A, 3-B, and 3-C). Reductions had been done on November 17 and December 1, 1944; two reductions were done on December 3; and an attempt at re-reduction was made on December 11. In desperation, the surgeon finally accepted the bad situation. The cast was still in place when the patient was first seen by the author, four months later. Arthrodesis of the ankle joint was done for disability and

relief of pain. From this, one can see the importance of recognizing the occasional case with posterior displacement of the fibula behind the tibia.

Two other similar cases of fracture-dislocation of the ankle, with posterior fixation of the fibula behind the tibia, have been seen. One patient, a woman, continued under conservative care, with the talus displaced laterally under the tibia, for two years. The malleoli became united with the tibia and fibula, but traumatic arthritic changes occurred

FIG. 4-A

Normal relationship of fibula and tibia, as shown by photographs and roentgenograms.

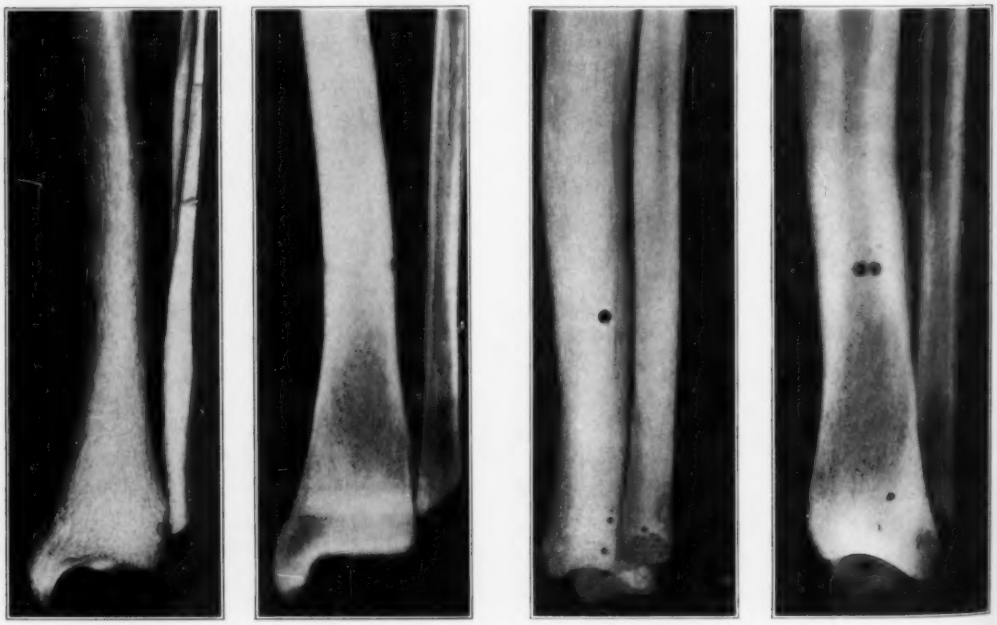

FIG. 4-B

Proximal fibular fragment has been displaced behind the posterolateral ridge of tibia (shown in photograph). Roentgenograms show no change from the normal on the anteroposterior view, and but slight change on the lateral view.

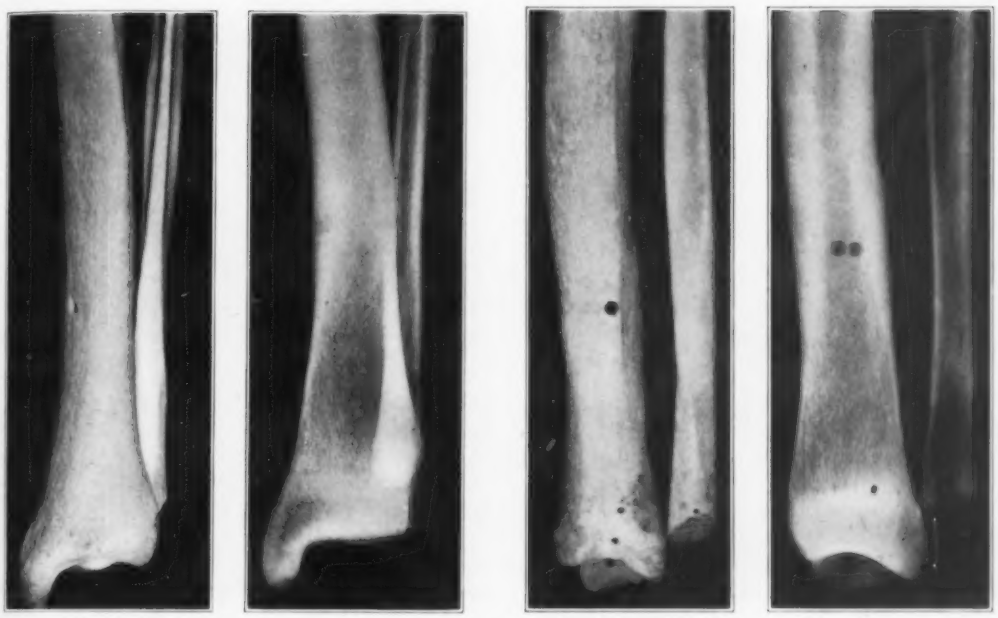

FIG. 4-C

Roentgenograms confirm the photographic findings of marked posterior displacement of the fibula behind the tibia. With the osseous structures of the foot and the lateral malleolus in place, faulty roentgenographic interpretation is generally made, even in this grossly displaced situation, because the interpreter believes the picture was taken in rotation or at an angle.

in the ankle joint and pain persisted. Two years after the fracture, the patient was seen by the author and ankle fusion was performed.

A police officer, who was seen nine months after injury, had union of the fibular fracture and widening of the tibial mortise. A diagnosis was made of posterior fixation of the fibula behind the tibia; at operation, this diagnosis was confirmed. The widening of the tibial mortise could not be overcome, even with the lateral malleolus separated entirely from the fibula above and from all other structures except the collateral ligament, until the proximal fragment had been displaced from behind the tibia and returned to its normal location. This was apparently due to the traction attachment of soft tissues from the talus and calcaneus to the proximal fragment of the fibula. Since the patient was an active and youthful person, reconstruction of the ankle mortise was attempted by simple fixation of the fibular fragments with screws. Six months after the operation, this patient returned to light police duty and has no demonstrable defects in or about the region of his damaged ankle.

The author believes that the situation described arises as follows: As the foot twists under the talus, with the leg continuing to push forward and to rotate outward, the lateral collateral ligaments draw the intact fibula behind the tibia. Continuation of the force rotating the talus backward and out from its position beneath the tibia causes further force on the lateral collateral ligaments; finally, the fibula is broken off against the posterior tibial border, as one would break a bone over a wedge in doing an osteoclasis. Following the fracture and the cessation of the forces, there is nothing left attached to the upper fragment of the fibula by which it can be drawn back into place. One may tear off the periosteum and swing the talus under, or nearly under, the tibia. It is impossible, however, to force the upper fibular fragment back around the posterolateral ridge on the lower tibia, because of the tight pull of the still intact interosseous membrane. Only by insertion of a pry between the two bones will enough leverage be produced to replace the fibula in its proper location on the lateral surface of the tibia. Upon inserting such a pry,

one will find that it passes transversely between the two bones instead of in an anteroposterior direction, because of the displaced osseous structures. A great amount of force is necessary for replacement, even with a pry. The reason all such fractures do not have the upper fragment of the fibula impacted behind the tibia is that, in most of them, the fibula breaks off (due to leverage against the lateral surface of the tibia) before it has been swung backward over the posterolateral tibial ridge. The interosseous membrane is undoubtedly too tough and tight in most instances to allow displacement of the fibula to occur before the fracture takes place.

Photographs and roentgenograms of an articulated specimen of a tibia and a fibula show that, on the lateral view in normal position, the tibia and fibula become superimposed and lie almost in the same straight line (Fig. 4-A). It has always been felt, in interpreting roentgenograms, that the fibula would be at a posterior level, because the medial malleolus is anterior to the lateral malleolus. This is not so, since the medial malleolus really arises from the front half of the medial surface of the tibia. When the fibula is slightly displaced and caught behind the posterolateral ridge only (Fig. 4-B), on the anteroposterior view it appears to be in its normal position on the lateral surface of the tibia. The lateral views, both in the photographs and the roentgenograms, show that the fibula is truly caught behind the lateral ridge of the distal portion of the tibia. Oftentimes roentgenograms are interpreted as showing no displacement of this upper fibular fragment, because it is believed by the roentgenologist that the view taken was not truly lateral or

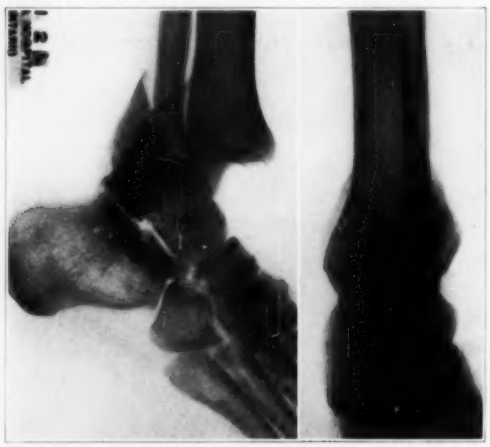

FIG. 1-A (Harris)

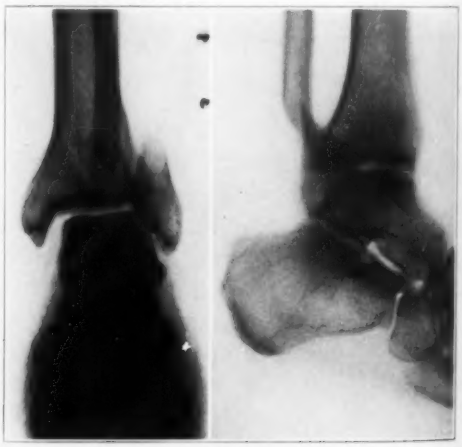

FIG. 1-B (Harris)

FIG. 1-C (Harris)

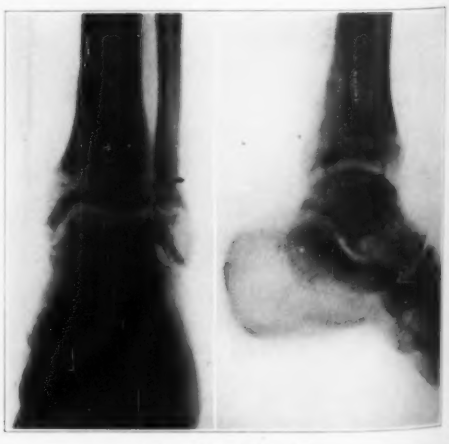

FIG. 1-D (Harris)

truly anteroposterior. In the badly displaced upper fibular fragment (Fig. 4-C), there should be no difficulty in interpreting the roentgenograms properly, if the roentgenologist understands that posterior fixation of the fibula behind the tibia can occur in fracture-dislocation of the ankle. Actually, the interpretation is generally wrong, because the possibility of such a situation has not occurred to the roentgenologist.

Having these facts in mind, if one reviews the roentgenograms for the first three cases presented here, it will be noted in each instance that gross discrepancies appear in the roentgenograms, pathognomonic of posterior dislocation of the fibula behind the tibia, and that in no preoperative roentgenogram shown was the fibula clearly seen to be in its normal relationship to the tibia.

SUMMARY

In fracture-dislocation of the ankle, one should make sure that the upper fragment of the fibula is not caught behind the posterolateral ridge of the tibia. In some instances, fracture of the lateral malleolus does not occur until the foot has twisted beneath the ankle joint, drawing the fibula behind the tibia and breaking it off against the posterior margin thereof. When this occurs, the fibula is firmly caught and needs open reduction with a pry; severe disability is likely to occur unless accurate replacement is made by operative means.

DISCUSSION

DR. R. I. HARRIS, TORONTO, ONTARIO, CANADA: A recent experience, fresh in my memory, is clarified by Dr. Bosworth's presentation. It can best be presented by illustrations. Figure 1-A shows the fracture-dislocation sustained by a farmer, forty-seven years old. In Figure 1-B is seen the result obtained by manipulations. Figure 1-C shows the best result which could be obtained by repeated manipulations.

Open operation was undertaken. The lower end of the upper fibular fragment was deeply buried behind the tibia. It was fixed firmly in this position, and strongly resisted replacement. Unlike Dr. Bosworth, we did not detect the true state of affairs. Strong leverage finally shifted the fragment, which then suddenly snapped back into something like its normal position. It was secured to the lower fragment by a suture of stainless-steel wire. The result of the operation is shown in Figure 1-D.

I now recognize that this difficult fracture problem was of the type described by Dr. Bosworth. It is well that it should be recorded, for I feel certain that his observations will explain hitherto unsatisfactory results.

THE FORMATION AND SIGNIFICANCE OF VERTEBRAL ANKYLOSIS IN TUBERCULOUS SPINES

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In reviewing the cases of tuberculous spondylitis at this Clinic, the author noted that in a few cases the clinical healing of the process coincided with the roentgenographic appearance of a spontaneous fusion of the anterior spine. In the cases where a prolonged roentgenographic follow-up was available, it was observed that these vertebral synosteoses, or vertebral blocks, had been formed either by end-to-end contact of two or more vertebral bodies after the destruction of the intervertebral disc and cartilaginous plates, or by recalcification and union of the remnants of several vertebral bodies which had been partially destroyed during the early stages of the disease.

The amount of concurrent deformity of the spine, and the shape, structure, and speed of formation of these synosteoses, were not uniform. In some cases, they appeared in only slightly deformed spines, and after periods of from two to three years; while in others, the fusion of the anterior spine took place when a marked kyphosis was already present, the formation of the block requiring a much longer period of time.

Interest in these vertebral blocks was aroused by the fact that the best clinical results in cases of tuberculous spondylitis were observed in the cases which healed with a minimum amount of deformity of the spine and with the formation of an osseous block which included all the infected vertebral bodies. Follow-up of these cases, for long periods of time after the clinical signs of activity had disappeared, failed to show either recurrence of the disease or increase of the deformity. Consequently, a study was made of the conditions under which the blocks were formed, to determine if they had any relationship to the age of the patient, the location of the lesion, or the number of bodies involved. From a practical point of view, especial interest concerned the possibilities of obtaining the blocks at will, either by conservative or surgical procedures.

Careful study of the spines which healed by the formation of vertebral synosteoses showed that three main factors regulated the speed of their formation and final configuration: the topographical distribution of the infection, the pathomechanics of the different spinal segments, and the treatment.

A. *Types of Tuberculous Spondylitis*

The types of tuberculous spondylitis vary according to the topographical localization of the primary focus. They have been given several names: Kaufmann described a cortical and a central tuberculous spondylitis; Loeffler spoke of a "spondylitis tuberculosa anterior superficialis" and of a "spondylitis tuberculosa anterior profunda"; Ménard divided the types into "cavernous" and "intervertebral"; Steindler described the anterior peripheral, the central, and the epiphyseal forms; Calvé and Galland described the group of the "osteites vertébrales centrosomatiques". Finally, Seddon introduced the term of "metaphysial tuberculosis" and Hellstadius that of "paradiscal tuberculosis".

In spite of the confusion produced by differences in terminology, it is evident that nearly all the writers agree that there is a definite relationship between the localization of the primary focus and the ultimate characteristics of the spondylitic process. The explanations of the variation in the site of the primary focus have been based on the studies of Lexer and his associates of the blood supply of the vertebral bodies. This subject has more recently been reviewed by Wagoner and Pendergrass. The different sources of blood supply

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to the different parts of a vertebral body will explain the different topographical types, which, in spite of the complex variety of names suggested, may be basically reduced to three: anterior, central, and epiphyseal.

In recent years, several authors (including Schmorl, Seddon, Junghanns, and Doub and Badgley) have taken exception to the term "epiphyseal tuberculosis", and several other terms have been proposed as substitutes. There is no doubt that the term "epiphyseal" is incorrectly used in most of the cases; because this particular type of tuberculous spondylitis appears frequently in adults after the secondary centers of ossification of the vertebral bodies have already fused, and very seldom is the process confined to the so-called vertebral epiphysis. On the other hand, the recent studies of Schmorl and Junghanns tend to deny that the secondary centers of vertebral ossification are true epiphyses. For these reasons, the term "metaphysial tuberculosis" (Seddon) or that of "paradiscal tuberculosis" (Hellstadius), is more in accord with our present knowledge of anatomy. This latter term will be used frequently, because it does not infer the accepted existence of a true vertebral epiphysis, meaning only a tuberculous process localized at the portion of the vertebral body contiguous to the intervertebral disc.

B. *Pathomechanics of the Tuberculous Spine*

In order to understand the final configuration and the mechanism of formation of the vertebral blocks, it is necessary to take into consideration the pathomechanics of the different spinal segments.

Due to the fact that, in tuberculous spondylitis, new-bone formation is as a rule nonexistent or very slight, the formation of a vertebral synosteosis is possible, in the great majority of cases, only after the approximation and superimposition of the affected vertebrae have taken place. This approximation can be performed either along a longitudinal axis (telescoping), by flexion in the sagittal plane of one spinal segment upon the other (called "inflexion" by Ménard), or, in a minimum number of cases, by flexion in the frontal plane (the so-called tuberculous scoliosis).

As described in detail first by Ménard and more recently by Calvé and Galland, due to some anatomical peculiarities, the amount of telescoping and "inflexion" that may take place between two vertebral bodies varies in the different segments of the spine. In the lumbar region, because of the size of the discs, the vertical position of the articular facets, and the relative narrowness of the pedicles, a marked amount of telescoping may take place. Conversely, the telescoping is minimum at the thoracic segment. In the cervical spine, the interposition of the transverse processes, after the destruction of the vertebral bodies has taken place, interferes with the telescoping.

As far as the tendency towards forward flexion is concerned, the maximum possibilities are found in the thoracic spine. In this segment, the loss of the nucleus pulposus produces a posterior displacement of the center of motion between the two vertebrae, a subluxation at the level of the articular processes, and an increase of the amount of body weight to be supported by the anterior part of the vertebral body. Later, if a complete destruction of one or more vertebral bodies has taken place, the orientation of the thoracic articular facets will allow the backward and downward displacement of those neural arches that have lost their connections with the anterior spine, thus producing a still further increase of the deformity.

None of these peculiarities is present in the cervical and lumbar segments where, as a rule, the deformities are much less pronounced. In the cervical segment, especially, the "inflexion" is always incomplete, because of the interposition of the transverse processes.

However, these regional characteristics alone cannot explain the differences in the final configuration of the different vertebral blocks. There are several other factors that should be taken into consideration; one of these is the decrease in the resistance of the body of the vertebra to pressure stresses, secondary to some types of tuberculous spondylitis.

In the so-called epiphyseal or paradiscal tuberculous spondylitis, the process appears on roentgenograms either as a progressive thinning of the intervertebral disc, with or without the presence of a circumscribed focus of osteitis, localized near the intervertebral space, or as a destructive process, extending all along the paradiscal region of one or two contiguous vertebral bodies. The rest of the vertebral body preserves its normal shape and structure, except for slight signs of decalcification. In the central types of tuberculous spondylitis, where the body as a whole presents signs of marked decalcification and softening, different phenomena occur.

In the first group of cases described, the resistance to pressure stresses of the infected vertebral bodies was not greatly diminished. In all the cases of tuberculous epiphyseal spondylitis, the patients had been walking and working for one or more years before seeking treatment, and in none was a collapse of the vertebral bodies observed. On the other hand, among the cases of central spondylitis, a massive flattening of one or more vertebral bodies was prone to occur, even while the patients remained in recumbency.

Another factor that influences the final shape of the vertebral synosteosis is the appearance of the protective muscle spasm; from the onset, it produces a complete flattening of the lordosis in the lumbar spine, thus allowing a uniform telescoping of the vertebral bodies in the cases of the epiphyseal type.

Finally, the amount of destruction that takes place in each body, the number of affected bodies, and the type of treatment are factors in the final configuration of the vertebral blocks. These factors will be described in detail in the study of the clinical cases.

From the study of this series of cases, no conclusions were reached regarding the relationship between the final shape of the synosteosis and the different pathological types of tuberculous spondylitis (productive, caseous, fibrocaseous, *et cetera*).

C. *Treatment*

The evaluation of the effects of the treatment upon the speed of the formation and final configuration of the different types of vertebral synosteosis was made through the roentgenographic study of our clinical cases. From them we selected only those that fulfilled the following conditions:

1. Cases which had had roentgenographic check-up every three months, not only during the active stage of the process, but for several years after a clinical cure had been obtained;

2. Cases that had healed by the formation of a synosteosis on the anterior spine;

3. Cases of tuberculous spondylitis proved by guinea-pig inoculation of pus obtained from an abscess; or, in the cases without superficial abscesses, patients with a typical clinical and roentgenographic process, plus a proved tuberculosis of some other part of the body;

4. Cases without secondary infections or sinus tracts.

Sixty-one spondylitic lesions fulfilled the above requirements. Cases of adult and infantile tuberculosis were studied separately. More detailed data about localization, age of the patients, and clinical characteristics, will be given in the discussion of each of the different groups.

TUBERCULOUS SPONDYLITIS IN ADULTS

1. *Formation of Vertebral Synosteoses in Cases of so-called Epiphyseal Spondylitis*

Twenty-five spondylitic foci were found. In twenty of them the tuberculous process was proved by positive guinea-pig inoculations. In the remaining five, the possibility of any other type of infection was ruled out repeatedly by the clinical and roentgenographic progress, agglutination tests, blood cultures, *et cetera*. In addition, each of these patients had an associated proved tuberculosis in some other region of the body.

The lumbar region was involved seventeen times; the thoracic spine in five cases; the

Fig. 1

Fig. 2

Fig. 3

Fig. 1: A case of tuberculous spondylitis proved by guinea-pig inoculation. Roentgenogram shows direct superimposition of two vertebral bodies after a slow and progressive thinning of the intervertebral disc for four years. No destructive osseous focus can be detected.

Fig. 2: Partial osseous union, localized at the anterior part of the vertebral bodies. As a space between the two vertebrae can still be observed, the destructive osseous focus was localized at the antero-inferior margin of the more cephalic vertebrae. The union was principally formed by peripheral new-bone formation and took place two and one-half years after the onset of the clinical symptoms. Patient had a proved tuberculosis of the kidneys and epididymis.

Fig. 3: Partial osseous union, occupying more than half of the intervertebral space. The posterior part of the intervertebral space is still visible. Original destructive focus was localized at the upper anterior region of the caudal vertebra. Patient had a proved tuberculous spondylitis, associated with pulmonary tuberculosis.

thoracolumbar junction in three cases. The average age of the patient at the time of onset was twenty-six, the youngest being nineteen and the oldest thirty-seven. Associated tuberculosis of the lungs was present in fourteen. The pulmonary process began two to three years before the spinal symptoms in six cases; in one it started six years before. In the remaining cases, the pulmonary involvement was discovered in the routine chest plates, taken in the examination of every case of suspected osseous tuberculosis.

In twenty cases there was a subcutaneous abscess, this being the first clinical sign in three of them.

Except for those that were already in recumbency for some other type of tuberculosis, most of the patients were seen for the first time one or two years after the onset of the clinical symptoms. None had had any previous treatment, and all had been walking and working up to the time of admission.

All these patients were treated by prolonged periods of conservative treatment. Following this, if the general condition, roentgenograms, and laboratory tests showed that the condition was in the healing stage, and no other active tuberculous focus was present in another part of the body, a fusion of the spine was performed. The technique used was the one described by Steindler. Following the surgical fusion, the patient remained in a plaster bed from three to four months, following which he was allowed to get up with a body cast for about three months, and finally a body brace was applied.

This series does not include cases treated exclusively by an early surgical fusion of the posterior spine. However, there were fifteen cases which were treated only by conservative measures, due to the presence of some other active extraspinal tuberculous focus, which necessitated keeping the patient in recumbency. During this time, a spontaneous fusion of the anterior spine occurred, and a surgical intervention was not performed.

Roentgenographically, three groups were most frequently seen:

First, there were cases characterized by a progressive and slow thinning of one intervertebral space. This thinning was usually accompanied by a subcutaneous abscess, and after three to four years the two vertebral bodies became directly superimposed (Fig. 1). The outlines and structure of the vertebral bodies remained very distinct throughout the duration of the process; and, in spite of the close contact, no osseous trabeculae running from one vertebral body to another were seen. These unions were considered to be fibrous unions.

Secondly, there were the cases characterized by the presence of a small destructive focus, localized immediately below or above the intervertebral space. If in the roentgenogram this focus appeared in the center of the body, the evolution of the process was usually identical to those just described. However, if the anteroposterior or lateral view showed that this destructive area was localized near the margins, after a period of thinning of the disc lasting between one and two years, a partial and osseous synosteosis between the vertebrae appeared (Figs. 2 and 3). As a rule, after this synosteosis had taken place, the remaining intervertebral space was spared. These unions were classified as partial osseous blocks.

Finally, there were the cases in which the destructive process was extended all along the paradiscal region of one or two vertebral bodies. The destruction of the intervertebral disc, as observed in the roentgenograms, was slow but complete; the two vertebrae became approximated by telescoping and the end result was a total and osseous union of two vertebral bodies (Figs. 4-A and 4-B). In some of these cases, the process started in one body and spread to the contiguous one; in others, the process started simultaneously in both of them, a phenomenon which can be explained on the basis of the characteristics of the arterial circulation. As demonstrated by Hanson and later by Solotuchin, the same branch of the posterior vertebral artery supplies the apposing surfaces of two contiguous vertebral bodies.

Peripheral new-bone formation was observed in only four cases without sinuses,

FIG. 4-A

FIG. 4-B

Complete osseous synosteosis of two vertebral bodies.

proved by guinea-pig inoculation. It appeared not earlier than one year after the onset of the clinical signs.

We were interested in knowing how much a posterior spinal fusion influenced the development of these blocks, especially from the point of view of accelerating their formation. For this purpose, the cases treated by prolonged recumbency alone and those treated by shorter periods of recumbency plus posterior surgical fusion, were compared. The conclusions obtained were as follows: (1) Independent of the type of treatment used, the roentgenogram showed that the type of vertebral block was related to the rapidity with which it was formed; (2) the surgical fusion of the posterior spine did not accelerate the formation of a vertebral block.

Under conservative or surgical treatment, the most rapid formation of vertebral synosteoses took place in the cases with circumscribed foci, localized near the margins of the vertebral bodies. Such a focus produced a partial osseous synosteosis in an average time of one and one-half years after the onset of the clinical symptoms. Next were the cases presenting a total destruction of the paradiscal regions; in these cases osseous ankylosis was complete in an average of three to four years after the onset of the clinical symptoms. Finally, in the cases characterized by a progressive thinning of the intervertebral space, fibrous union was obtained in from four to five years. The eventual transformation of this fibrous union into osseous synosteosis was observed in two cases, seven years after the onset of the clinical symptoms.

Some writers (Hibbs, Albee, Allison and Hagan) emphasize the point that a surgical fusion of the posterior spine should be performed as early as possible, in order to obtain the best result in tuberculous spondylitis. In our series there was only one case fused during the early stages; therefore, we are unable to confirm or deny this point. However, it is interesting to note that several authors (Calvé, McKee, Seddon) believe that the early application of an osseous graft will interfere with the future formation of the vertebral synosteosis.

From our experience, it may be stated that, if a posterior spinal fusion is performed when the process is in the healing stage and there is only a very small gap between the two involved vertebrae, it does not interfere with the final formation of a block. In other words, if the general condition of the patient allows him to be ambulatory and there are no other contra-indications arising from the vertebral lesion, it is not necessary to prolong

the recumbent period for completion of the vertebral block. The posterior surgical fusion can be used in these cases as a means of increasing the spinal stability, and thus obtaining a better protection during the first months of resumed activities. However, it is interesting to remember that, as Seddon obtains similar results by the use of a body brace, the surgical fusion should not be considered essential and should be used only in the cases in which, due to the localization of the process, complete immobilization cannot be obtained with a body brace.

The prognosis of these blocks was excellent, regardless of type. Not only was the amount of spine deformity minimal, but observations over periods ranging from two to ten years after the clinical arrest of the process, failed to show signs of collapse or reactivation of the process. This was true in the cases surgically fused, as well as in the cases treated conservatively throughout.

2. *Formation of Vertebral Synosteoses in Cases of Central Tuberculous Spondylitis*

The tuberculous infection may spread inside the vertebral body, producing a diffuse tuberculous osteomyelitis. In some cases observed during the early stages of the disease, the lesion was clearly localized at the center of the vertebral body, and later produced a secondary involvement of one of the intervertebral spaces. However, in most of the cases seen several months or years after the onset of the clinical signs, it was impossible to ascertain the localization of the primary focus. In only two cases of this group of central spondylitis did the process start as a progressive thinning of the intervertebral disc. They differed from the ones belonging to the group of epiphyseal spondylitis, in that the disappearance of the intervertebral space, as seen in the roentgenogram, was complete six months after the onset of the clinical symptoms.

Fifteen spondylitic lesions which fulfilled all the requirements already described were included in this study. Three of them were proved cases of tuberculosis by guinea-pig inoculation of pus obtained from a paravertebral abscess. There was an active tuberculosis of the lungs in ten cases, of the kidney in two, and of the hip in one.

The age of the patients was between seventeen and fifty-three, the average being twenty-six years. The lesion was localized in the lumbar spine in four cases; and in the thoracic segment, in eleven cases. In all of these eleven thoracic cases, abscesses were evident in the roentgenograms. None of these abscesses became superficial.

In five cases the pulmonary symptoms started two years before the spinal ones. In one case, the pulmonary infection was discovered two months prior to the spondylitic lesion. In the others, the lesions were found during the routine check-up on admission.

In all cases the original lesion was confined to two vertebral bodies, with total destruction of their corresponding intervertebral spaces. In only two cases did the process invade two additional vertebral bodies before reaching the healing stage. Widespread destruction of multiple vertebral bodies and intervertebral spaces was not observed, even in the cases in which there was a secondary anterior involvement of several vertebrae above and below the primary focus.

There were several factors that made the formation of a vertebral block somewhat different in these cases from those belonging to the group of paradiscal spondylitis: First, the roentgenographic disappearance of the intervertebral space was a rapid phenomenon, as was also the spreading of the infection into the two contiguous vertebral bodies. Second, due to the more intense destruction of the vertebral bodies, the appearance of wide gaps between the spinal segments was frequently observed. Third, the intrinsic resistance of the vertebral bodies was diminished in such a way that the phenomenon of sudden collapse was prone to appear. Fourth, anterior involvement of several bodies above and below the primary focus was frequently seen. Fifth, central or peripheral new-bone formation, intense enough to fill the gap between the remnants of the vertebral bodies, after the destructive period was over, was never observed; and, consequently, the formation of a block

FIG. 5

FIG. 6

Fig. 5: New vertebral body, formed by the remnants of two affected vertebrae. This patient had tuberculosis of the spine, associated with tuberculosis of the lungs.
Fig. 6: Synosteosis of two vertebral bodies in a healed case of proved tuberculosis of the spine.

depended upon the ability of the involved spinal segment to obtain an approximation between the partially destroyed vertebral bodies.

Taking all these differences into consideration, we may summarize the formation of these blocks as follows:

When the destruction of the anterior walls of the infected bodies was not marked, the gap formed during the destructive stage was predominantly obliterated, either through telescoping or forward flexion, according to which one of the spinal segments was involved. Later on, a new vertebral body appeared, formed by the fusion of the vertebrae which had been partially destroyed. The new synosteosis had an anterior wall, sometimes only one-half or one-third the size of that of a normal vertebra (Figs. 5 and 6).

If the destruction of the anterior wall of both infected vertebral bodies had been far advanced or complete, the anterior involvement of several vertebral bodies situated above and below the primary infected vertebrae was frequently observed. The forward flexion of the upper spinal segment started during the early stages of the process. In the thoracic and thoracolumbar segments, the process continued unchecked until contact was made anteriorly between the upper articular surface of the more cephalically placed vertebral body and the lower articular surface of the body of the more caudally situated vertebra, thus producing the appearance of a triangle in the roentgenogram. As a rule, unless these patients were kept in recumbency for very long periods of time, the deformity increased, and two more contiguous vertebral bodies became involved. In the lumbar segment, the tendency toward forward flexion and kyphos formation was much less pronounced.

There were some exceptions to this general development. Some of the cases were similar to those described by Hellstadius under the name of "vesicular type" of tuberculous spondylitis; others were characterized by a slow and progressive destruction of the fourth lumbar vertebral body and intervertebral disc, with slight erosion of the upper articular surface of the fifth lumbar. In general, these cases followed more or less the pattern observed in the epiphyseal type of spondylitis.

In all of the cases of central spondylitis which healed by formation of a vertebral block, anterior ankylosis of the spine was obtained, while the patients were in recumbency. Follow-up from three to five years after the clinical healing of the process showed no

increase of the deformity, reactivation of the process, or phenomena of sudden collapse.

As far as spine deformity is concerned, the end results could have been much better in a high percentage of cases. One of the main reasons for the failure to control deformity was the inability to determine the exact moment when the patient should resume the standing position. As a rule, they were allowed to get up before the vertebral block was capable of withstanding daily stresses, sudden collapse sometimes occurring a few weeks afterward (Figs. 7-A and 7-B). For this reason, it was important to determine what roentgenographic signs differentiate an *unsound* vertebral block from one capable of preventing further increase of the deformity. The conclusion was reached that a vertebral block, appearing after a central spondylitis, cannot be considered *sound*, unless it fulfills the following requirements:

1. The outlines should be sharp and well defined. In the cases where a central cavity is present (Fig. 6), the outlines of this cavity should also be sharp and well circumscribed.

2. The density of the vertebral block, as shown by roentgenogram, should be equal to that of the surrounding non-affected vertebrae.

3. No signs of erosion should be present in either the upper or the lower articular surfaces of the newly formed vertebral body.

4. No paravertebral soft-tissue shadows should be present, especially if the lesion is localized in the thoracic segment.

In the thoracic and thoracolumbar segments, a block with all these characteristics was obtained only after from five to seven years of recumbent treatment. The question arises as to whether we are justified in keeping a patient in recumbency for such a long period of time, or if it is possible to obtain similar results in a shorter period by surgical fusion of the posterior spine.

The advantages and disadvantages of fusing a tuberculous spine have been discussed in detail by Steindler, and consequently only a brief résumé of our findings is necessary. Our conclusions are based on a comparative study of the end results in the patients fused after a vertebral block had been obtained and those fused and allowed to get up before an anterior synosteosis had occurred. The findings were as follows:

1. In cases of central spondylitis when the patient had been treated by recumbency

FIG. 7-A

FIG. 7-B

Fig. 7-A: A case of bilateral pulmonary tuberculosis and tuberculosis of one knee. The lateral view showed what appeared to be a solid block, formed by two partially destroyed vertebral bodies. The patient was allowed to get up with a brace two years after the onset of the spinal symptoms.

Fig. 7-B: Roentgenograms taken three months after the patient was allowed to get up showed a pathological fracture. She had not had any injury during the interval.

for periods of from three to five years, fused, and allowed to get up with a body cast three or four months after fusion, but before a sound vertebral block of the anterior spine had been obtained, the surgical fusion did not:

a. Produce the formation of a sound block;
b. Prevent further flexion of the spine;
c. Prevent the appearance of pathological fractures;
d. Prevent the spreading of the process to the surrounding vertebrae;
e. Prevent the appearance of new abscesses;
f. Prevent the appearance of neurological complications.

2. On the other hand, in the patients not allowed to get up until after a sound block had been obtained by conservative means, no signs of reactivation or collapse have been observed, whether or not fusion had been performed.

In the presence of these findings, it would be erroneous to conclude that the posterior fusion of the spine is of no value in the treatment of tuberculous spondylitis. This could be accepted, if a vertebral block could be obtained in every case of central tuberculous spondylitis. Unfortunately, due to the extension of the process, to its intensity, or to some peculiarities of the involved spinal segment, in some cases, an anterior synosteosis of the anterior spine does not take place, in spite of the use of very prolonged periods of recumbency. This occurs frequently in children, and in these cases the fusion of the posterior spine, while not ensuring against further increase of the process or of the deformity, increases the stability of a vertebral column, weakened by the process, by the "inflexion", and by the local subluxations.

In short, a sound vertebral block should be obtained every time that it is possible, even if it means prolonging the recumbency period; because it ensures a better prognosis, as far as the future stability of the spine and the healing of the process are concerned.

TUBERCULOUS SPONDYLITIS IN CHILDREN

While the general mechanism of block formation in children is similar to that observed in adults, the study of this series of cases revealed some peculiarities that warrant a further description.

In the first place, the division between epiphyseal and central types was not as definite; and, due probably to the size of the vertebral bodies, it was not infrequent to observe cases in which a primary epiphyseal infection extended far inside the vertebral body. Second, it was much more common to observe involvement of several contiguous vertebral bodies. Third, the number of cases in which a block could not be obtained was much higher in children than in adults. With the exception of two cases, no sound vertebral blocks were observed, if the tuberculous process involved more than five vertebral bodies. Fourth, in cases with localization to two or three vertebral bodies, the configuration of the block sometimes improved during the growing period.

The series of cases studied included twenty-one spondylitic foci in children, each of which healed by the formation of a vertebral block. These twenty-one cases were compared with several others of a similar type, in which no vertebral synosteosis had been obtained during recumbency.

Six of the cases that healed by formation of a block were cases of tuberculosis, proved by guinea-pig inoculation. Those remaining had a proved tuberculosis of some other part of the body. All were cases of spondylitis without sinuses.

In each of four cases, the process was confined to two vertebral bodies. Under conservative treatment, osseous synosteosis was obtained three years following the onset of the clinical symptoms. Observation, from three to seven years after clinical healing of the process had been obtained, showed no reactivation of the process or further collapse of the spine.

In three cases, the infection involved three vertebral bodies. The pattern of formation was quite uniform. After the acute stage was over—during which the centrally situated body practically disappeared—recalcification took place and two of the affected vertebrae united, forming a block which was unsound because it did not include all the diseased vertebral bodies. A sound block was obtained two or three years later, which included all the affected vertebral bodies (Figs. 8-A, 8-B, and 8-C). Observations from three to six years after resumption of activities demonstrated a complete healing of the process.

FIG. 8-A
Showing affected vertebrae.

In twelve cases, the process involved four or five contiguous vertebral bodies. In these cases the final shape of the block was directly related to the amount of compression and wedging suffered by the two or three vertebrae most centrally situated. The two vertebral bodies situated at the extremes of the row, while presenting phenomena of erosion of the articular surface facing the center of the row, retained their normal structure and configuration fairly well. In the cases in which weight-bearing was not allowed until the most centrally situated bodies presented signs of good recalcification, the spine deformity was not too marked. On the other hand, if the patients were allowed to get up before this recalcification and final block formation had taken place, whether or not a spine fusion had been done, the most centrally situated

FIG. 8-B

FIG. 8-C

Fig. 8-B: Spontaneous union of two vertebrae which proved unsound.
Fig. 8-C: A sound block, including all the affected vertebrae, was later obtained.

vertebral bodies were markedly crushed and destroyed. The process of collapse of the spine continued as a rule until the two most distally placed vertebral bodies established partial contact at the level of their anterior parts.

In five of this group of twelve cases, spine fusions were performed after the children had been in recumbency for five or six years, at which time an anterior vertebral block was already present. In six other cases, in which also vertebral blocks had been obtained by prolonged recumbency, no surgical intervention was performed. The remaining patient was treated by recumbency during only one year; fusion was done; and the child was allowed to get up five months later in a body cast. Roentgenographic check-up for several years showed this to be the only one of this series in which no block had been obtained, and in which the deformity continued to be progressive.

When the tuberculous process had affected more than five contiguous vertebral bodies, *sound* blocks were observed in only two cases. In both cases, the process had affected the whole lumbar spine and the last two thoracic vertebrae. Under conservative treatment, synosteoses, including all the diseased vertebrae, were produced.

In the other cases, no blocks could be observed. For comparative purposes, the progress of these cases for several years was observed. All had been under periods of conservative treatment for four or five years. In practically all cases, in spite of the use of hyperextension and traction, the deformity increased and the process continued to progress for several years. Later, after spine fusions, these patients were allowed to get up, and invariably the spine deformity became more accentuated.

Whether or not this marked destruction and deformity could have been prevented, either by combining fusion and recumbency or by performing a very early fusion of the spine, cannot be stated. However, it is at present our conclusion that the surgical fusion of the posterior spine is not capable of preventing an increase of the deformity when the anterior spine is mechanically unsound, because of the presence of gaps, marked destruction, or decrease of resistance of the vertebral bodies.

CONCLUSIONS

The effects of a given treatment upon the evolution of a tuberculous spondylitis cannot be properly evaluated, unless the different topographical types of tuberculous spondylitis, the number of infected vertebrae, the pathomechanics of the different spinal segments, and the alterations in the structure of the vertebral body produced by the infection are taken into consideration. Only after careful consideration of all these factors, is it possible to understand why in some cases sound healing may take place in one and a half to two years after the onset of symptoms, while in other cases the process continues to progress for longer periods of time.

NOTE: In regard to the incidence of formation of a vertebral synosteosis in cases of tuberculous spondylitis, no accurate percentage could be obtained from our material. This is due to the fact that we never considered suitable for purposes of *clinical investigation* those cases that, although clinically and roentgenographically typical of tuberculous spondylitis, did not fulfill the above described basic requirements. To state that those discarded cases were not tuberculous would be against the best clinical and roentgenographic judgment; to accept them as cases of tuberculosis would be against scientific accuracy. We considered this last point far more important than any other, and consequently renounced all statistical investigations.

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PRIMARY CONGENITAL SUBLUXATION OF THE HIP *

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In cases of congenital dislocation of the hip, Frenchmen, contrary to Anglo-Saxon authors, distinguish between luxation and subluxation. In the latter, according to recognized authorities, the head of the displaced femur remains in contact with a more or less deformed acetabulum.

The importance of this distinction is even greater than was formerly thought. My studies have shown that the primary subluxation of the hip presents some anatomical characteristics distinctly different from those of luxation. The use of arthrography, as practised by me since 1935, enables one to distinguish a subluxation from a luxation, without difficulty and even in the youngest child.

As pointed out by Gourdon as early as 1906, subluxation, thus defined, is a relatively stable condition which never tends to develop into a luxation.

The prognosis of this primary subluxation remains grave for a long time in most cases, because experience has shown that the results of closed reduction of subluxation, even in a young patient, are very seldom satisfactory. However, the reduction of the displacement—that is, the lowering of the head into the acetabulum—presents no difficulty in such a case. In spite of orthopaedic treatment, the articulation remains incongruent and, sooner or later, there appear signs of a deforming osteo-arthritis, which eventually becomes permanently disabling. The treatment of an established osteo-arthritis raises considerable controversy; all one can say is that surgeons have not yet discovered the means of effecting a real cure. In my opinion, the problem is the prevention of the deforming osteo-arthritis consequent to the subluxation of the hip. That is the principal subject of the present article.

At the start, I would specify that I am considering here only the primary congenital subluxation. The so-called "residual" subluxations, which are frequently observed after attempted bloodless reduction of true luxation of the hip, are quite different. They are characterized, in particular, by the presence of interposed soft parts (limbus, round ligament, and capsule) between the head and the acetabulum; such interposition never occurs in primary subluxation.

DIFFERENCES BETWEEN SUBLUXATION AND LUXATION

These differences have been established with the help of arthrography, and the findings have been verified in the course of open reduction by the intra-articular approach.

The essential difference between these two varieties of malformation is indicated by the position of the cartilaginous roof, formed by the union of the non-ossified part of the acetabular rim with the cotyloid fibrocartilage (also called the limbus). *In a subluxation the limbus is forced upward and inward toward the iliac fossa; in a luxation the limbus is forced downward and inward toward the acetabulum.*

Such a distinction is clearly seen in Figures 1-A, 1-B, and 1-C (this patient was selected from a number of different cases). In this girl, aged seven and one-half months, the ordinary roentgenogram does not show any difference between the dislocation of the hip on the right side and that on the left side. Arthrograms, on the contrary, prove that on the right side there exists a subluxation with forcing of the limbus toward the iliac fossa. On the left side, there is a luxation with forcing of the limbus toward the acetabulum, which gives the articular cavity the classic hourglass appearance.

There are, however, a whole series of other anatomical differences between subluxation

* Read at the Annual Meeting of The British Orthopaedic Association, in London, October 26, 1945.

and luxation. Most of these differences, which are outlined in the schematic drawings (Figs. 2-A and 2-B), may be readily discerned on arthrograms.

The Acetabulum

In *subluxation*, the cartilaginous roof and the limbus, forced by the head of the femur against the external iliac fossa, always appear atrophied. By this pressure, the cavity is worn down in its upper part and generally takes on a "scooped-out" appearance, which is characteristic. This compression and atrophy of the cartilaginous roof present one of the essential points to be kept in mind in a consideration of the treatment of a subluxation.

In *luxation*, on the other hand, the cartilaginous roof and the limbus are forced toward the acetabulum. In such a case the limbus is always more or less hypertrophied. The appearance of the hypertrophied limbus in the arthrograms contributes to the creation of a constriction of the articular capsule, known as the "hourglass contraction". Moreover, the hypertrophied limbus frequently remains interposed between the head and the acetabulum during attempts at closed reduction of the luxation,—a point upon which I cannot enlarge in this article, which deals only with subluxation. However, apart from the interposition of soft parts, the acetabulum in a luxation generally retains a satisfactory depth with a well-developed roof. This fact has already been observed in England by Fairbank, as a result of the examination of the pelvis of subjects suffering from congenital luxation, who had reached an advanced age without any attempt at reduction having been made.

The worn down or shallow acetabulum, therefore, is the characteristic of subluxation, and not of luxation, as is stated in orthopaedic textbooks.

The Head of the Femur

In *subluxation* the femoral head is deformed very early. It is enlarged, being widened transversely, as well as flattened at its supero-internal pole. This is shown in arthrograms of very young children, where the contour of the cartilage of the head, indicated by the contrasting medium, reveals this hypertrophy even when the osseous nucleus is poorly developed.

FIG. 1-A

Girl, seven months old. Roentgenogram does not clearly differentiate luxation from subluxation.

In *luxation*, on the other hand, the head retains a regular contour for a long time. As regards its volume, the head appears slightly smaller than normal in the majority of cases.

The Articular Capsule

In *subluxation* the capsule, although enlarged, is never interposed between the femoral head and the acetabulum. What is more, intra-articular operations have shown that the round ligament is practically always absent.

In *luxation*, on the other hand, the capsule often is interposed between the head and the acetabulum, especially at the lower portion of the cavity. The round ligament is present in about one third of the cases. It is often so big as to constitute an obstacle to reduction. The hypertrophied round ligament is always visible on arthrograms.

FIG. 1-B

Arthrograms show subluxation of the right hip and luxation of the left. Note the difference in the shape of the head in luxation and subluxation.

FIG. 1-C

After closed reduction, all the tests of a good reduction are present. Note that the tip of the roof reaches the Y line.

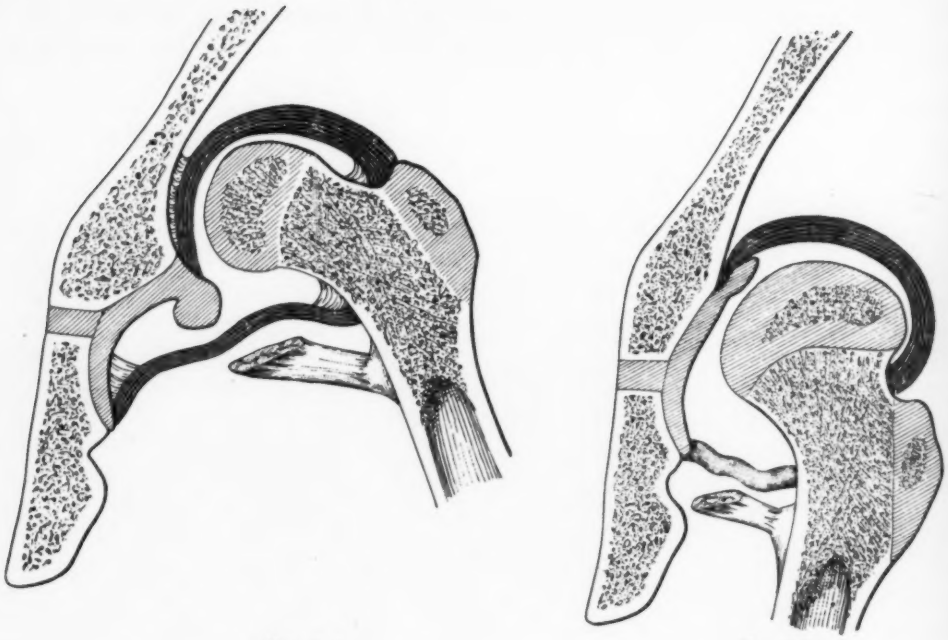

FIG. 2-A FIG. 2-B

Diagrams showing the essential differences between luxation and subluxation.

It is clear, therefore, that the interposition of soft parts (the limbus, the round ligament, and the lower fold of the capsule) is the rule in luxations, where it constitutes an obstacle to reduction. Such an interposition of soft parts never exists in subluxation.

The Neck of the Femur

In *subluxation* one often finds a valgus of the neck, the angle of which measures from

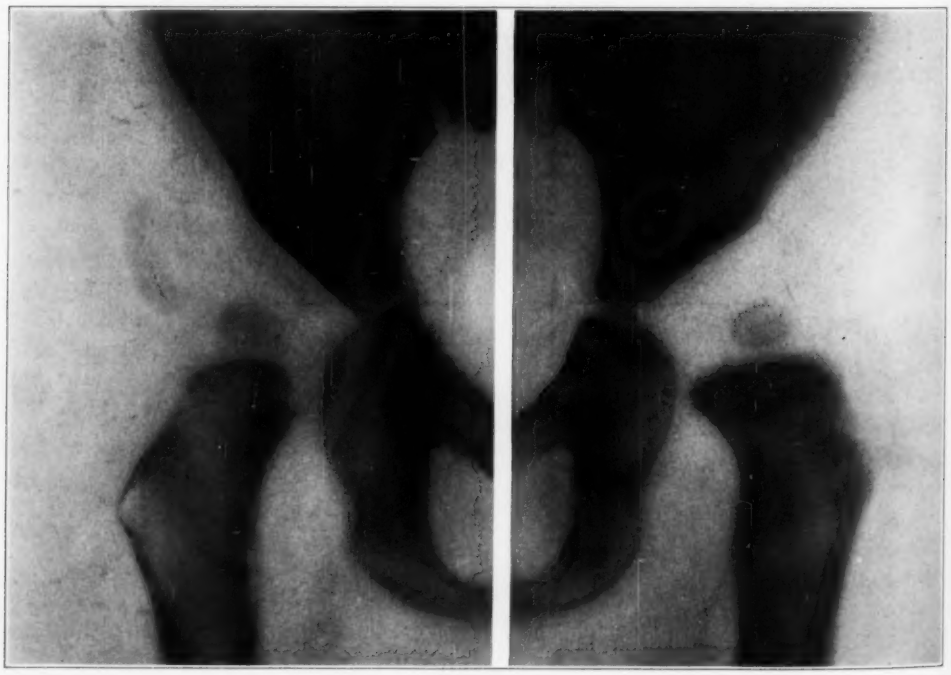

FIG. 3-A

Roentgenogram of a girl, twenty-one months old.

150 to 155 degrees, whereas on the sound side the angle measures 130 degrees. The presence of the valgus of the neck is indicated in the observations of the various authors, such as Klapp and Waldenström, who have described "coxa valga luxans". The anteversion of the femur, measured as exactly as possible by the methods of Perry Rogers, of Dreesmann, and of Netter, is very common in subluxations (the author has found it in 68 per cent.

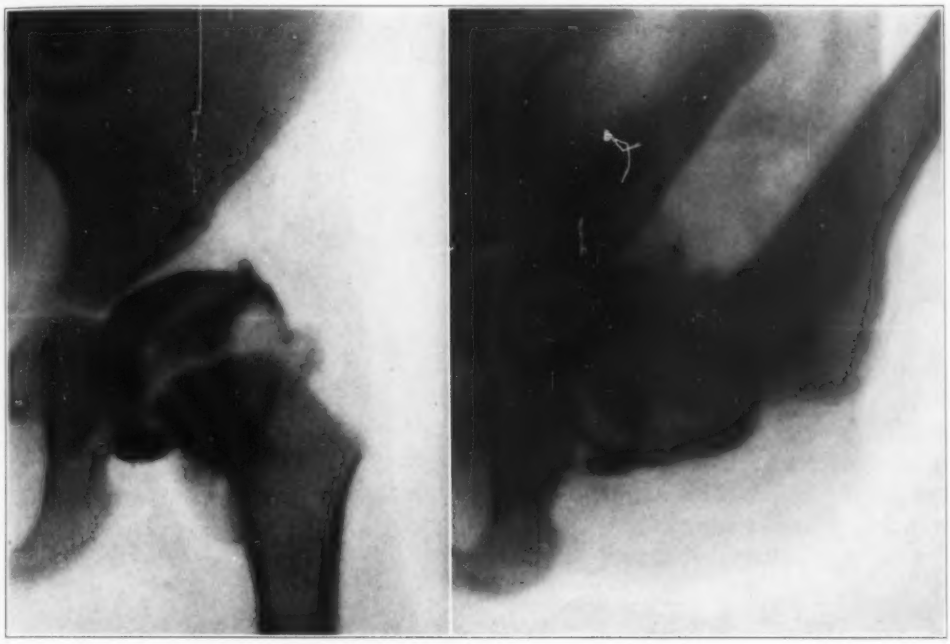

FIG. 3-B FIG. 3-C

Bilateral subluxation, evident in the arthrograms.

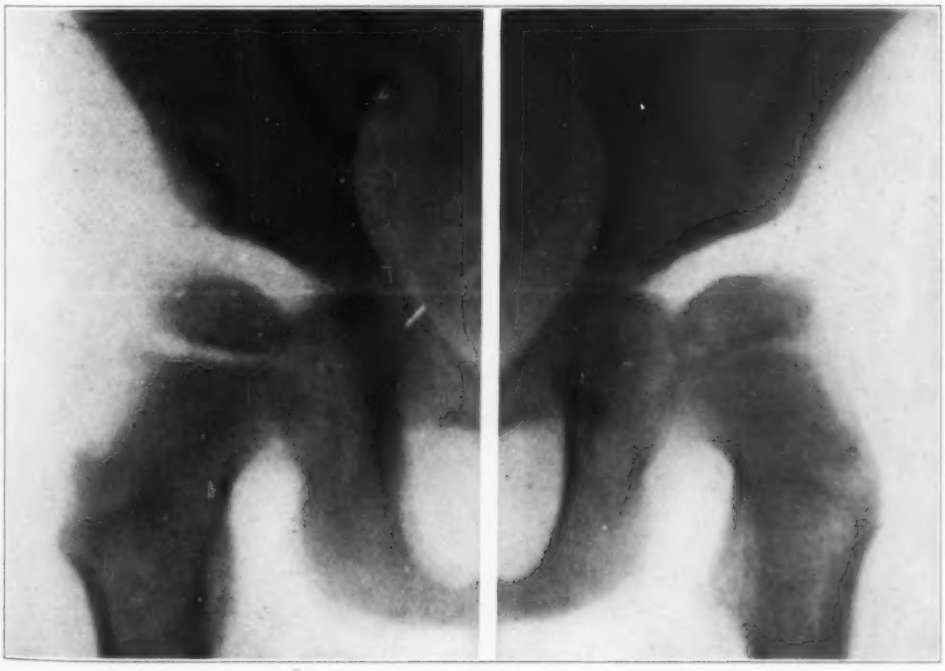

FIG. 3-D

Good result two years after closed reduction.

of his cases). This anteversion exists even in very young patients. It is therefore very early, if not primary. The degree of this anteversion undergoes practically no change in the course of life.

In *luxation,* on the contrary, there is no valgus of the neck. The anteversion is found only exceptionally in young subjects. In the course of life it increases progressively, and may reach a high degree in elderly subjects suffering from high luxation. In luxation, therefore, the anteversion seems to be secondary and late.

All these anatomical differences between subluxation and luxation are tabulated (Table I). They are easy to recognize and exist in the majority of cases.

In the course of our surgical experience, which is already considerable since it is based upon over 250 cases of luxation and subluxation, we have observed not more than two cases which may be exceptions to the rule,—that is, cases in which characteristics of both luxation and subluxation were found together.

It follows from my studies, therefore, that subluxation and luxation represent different malformations.

Developmentally it can be seen that, in luxation, the head may, in time, rise higher. My arthrographic studies have established that the proportion of high heads with narrow hourglass capsular constrictions increases with age, but we are still within the domain of luxation.

In subluxation, on the other hand, one may see the acetabulum wear down gradually or the head become deformed to a maximum degree, but the subluxation remains always a subluxation. In our opinion, not one well-established fact can prove that a subluxation, showing the characteristics which have been described, can become a luxation. Moreover, it is inconceivable that the limbus, forced upward and atrophied, as it is in a subluxation,

FIG. 4-A

Roentgenogram of a girl, seventeen months old, showing subluxation on the left side.

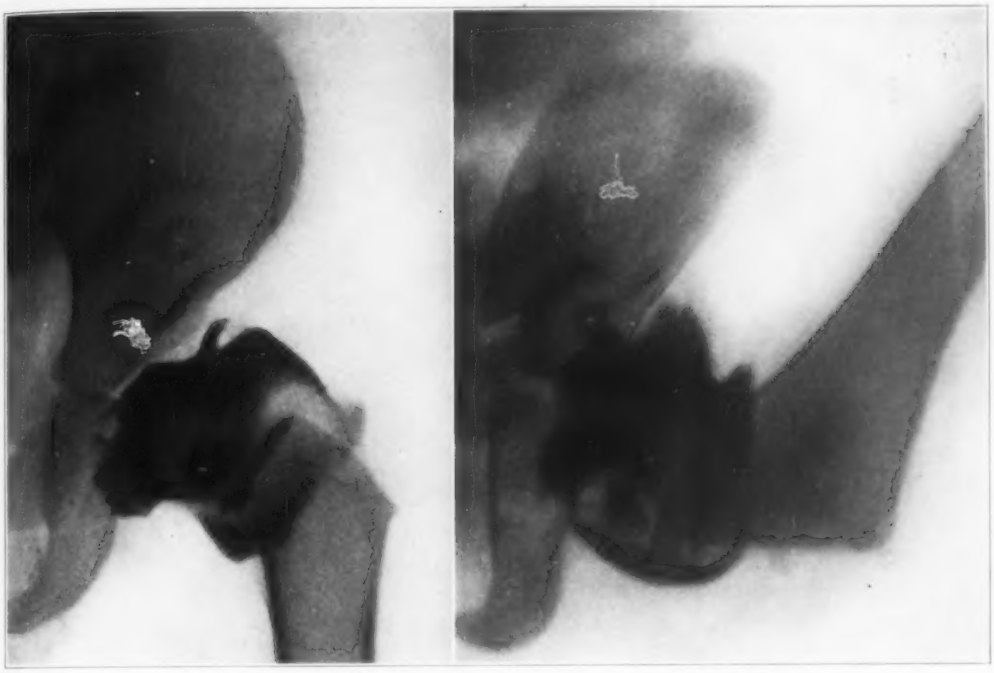

FIG. 4-B FIG. 4-C

Insufficient reduction, shown by arthrograms. Nevertheless, the treatment for closed reduction by the Lorenz method was continued.

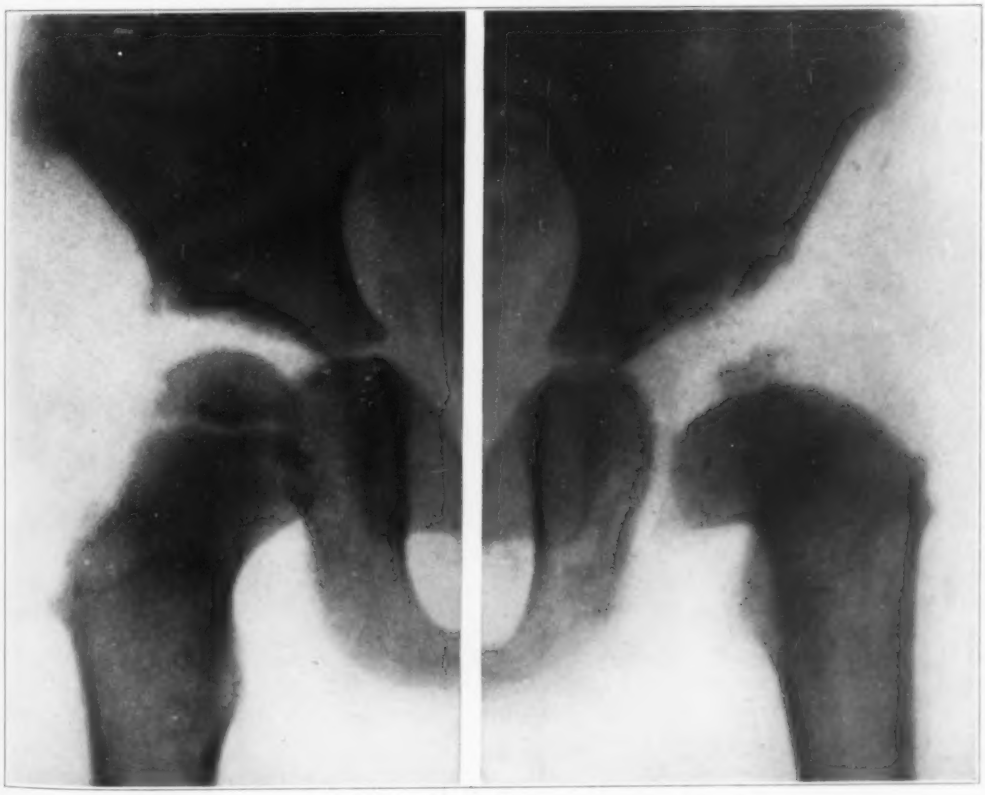

FIG. 4-D

Result after two years and eight months, showing poor reduction and osteochondritis of the head.

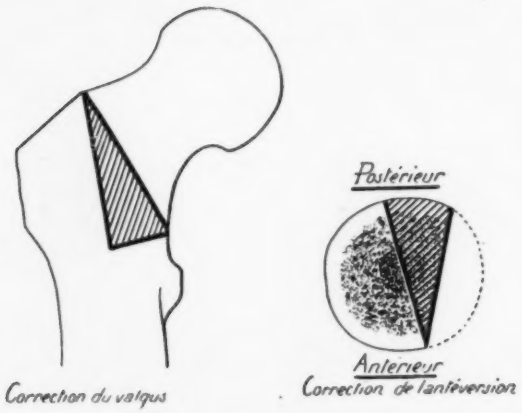

FIG. 5

Diagrams showing the Zahradníček method of correction of valgus and anteversion of the neck of the femur.

might in time become the limbus forced down toward the acetabulum and hypertrophied, as is seen in luxation. Still more is it inconceivable that the round ligament, which is absent in subluxation where the displacement of the head is very small, might reappear hypertrophied in a large number of luxations, even high luxations.

Without doubt subluxation and luxation have their origins in a common ground, since both malformations may coexist in one subject or may appear alternately in the course of hereditary transmission of the disease. However, both malformations originate at a stage of the embryonic development which it is impossible for us to determine, and under the influence of causes which are totally unknown to us. I can state, nevertheless, that from the initial stage, subluxation and luxation develop individually toward essentially different anatomical conditions.

CONCLUSIONS AS TO TREATMENT

The anatomical facts just described give an accurate idea of the difficulties which will confront the surgeon when dealing with a congenital subluxation of the hip,—that is, when he attempts to reconstruct a hip so that *the articular surfaces will be perfectly congruent.*

To achieve this condition is, in our opinion, the only way to prevent the appearance of a deforming osteo-arthritis, which is the almost inevitable consequence of an articular dislocation, imperfectly reduced.

I. *Closed Reduction*

The bloodless reduction must be checked rigorously by arthrography, which is done with a solution of *ténébryl* in a concentration of 30 per cent. This contrasting substance, identical with that used in urography, is rapidly eliminated—in about thirty minutes—without affecting the articulation. There is no need to emphasize the fact that only by arthrography can one visualize roentgenographically the state of the soft parts (cartilage of the roof, limbus, and capsule), of which hitherto one would have no idea.

The tests for a perfect reduction are the same as those indicated by Faber for a normal articulation:

(*a*) The tip of the limbus must come down in contact with a straight line drawn between the Y-shaped cartilage.

(*b*) The free edge of the cotyloid fibrocartilage must clasp a good half of the head.

(*c*) There must be no accumulation of the contrasting substance between the head and the center of the acetabulum.

In my experience the above conditions can only be fulfilled in very young children, less than two years of age, and even then, not in all cases. (This is the age at which we exceptionally see congenital subluxation of the hip.)

Experience has also shown us that the first Lorenz position gives a far better reduction than the position in abduction recommended by Putti.

Every time we left unchanged a manipulative reduction which the arthrograms showed to be unsatisfactory (the tip of the roof insufficiently lowered, the head incompletely clasped by the cotyloid fibrocartilage, and some contrasting substance remaining

between the head and the acetabulum), the results were bad. Not only was the reduction not effected, but in many cases there occurred an osteochondritis of the head which resulted in an even more serious deformity (Figs. 4-A, 4-B, 4-C, and 4-D).

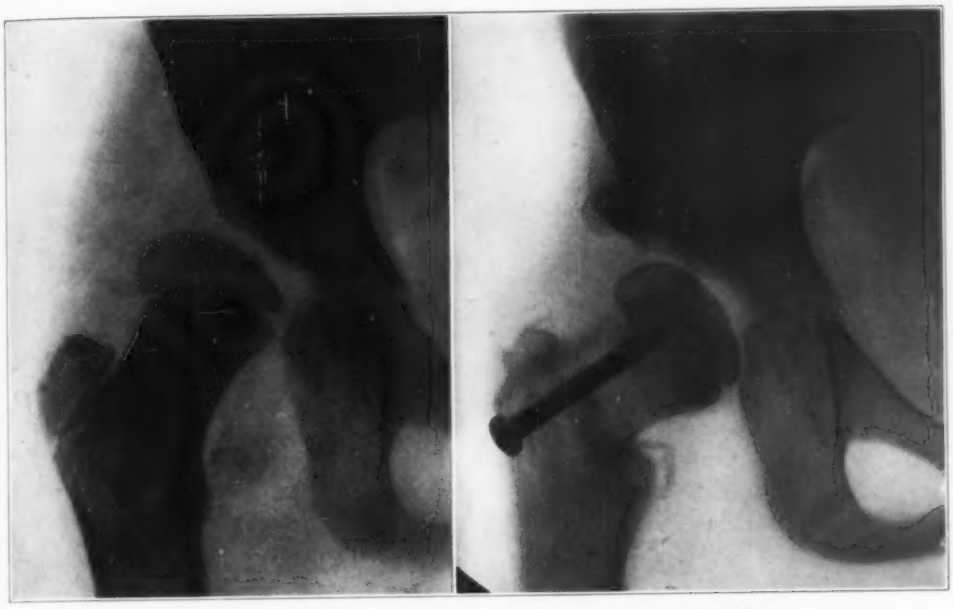

FIG. 6-A FIG. 6-B

Fig. 6-A: Roentgenogram (Nov. 1942) of a girl, seven years old, showing subluxation on the right side.

Fig. 6-B: Feb. 1943. After reduction by the extra-articular method.

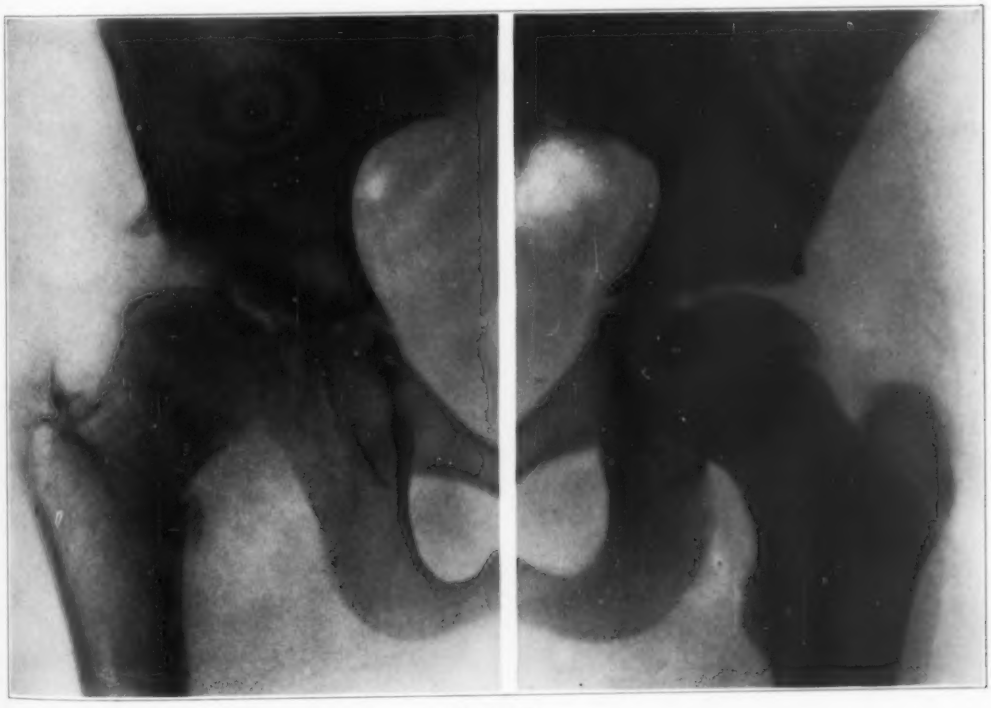

FIG. 6-C

June 1946. Good result four years after the operation.

II. *Construction of an Artificial Roof*

This purely palliative operation seems to us completely illogical. It fails to correct the articular incongruity. In addition, as Wiberg reported in two cases, a deforming arthritis of the head may develop in spite of a perfectly successful artificial roof.

III. *Open Reduction*

This is the only means left to reconstitute good articular congruity in most subluxations. The most favorable age for this operation is around three years. The operation can be performed on older subjects (fifteen years and over) provided that the components of the articulation have not been deformed, especially by untimely orthopaedic manipulation.

FIG. 7-A

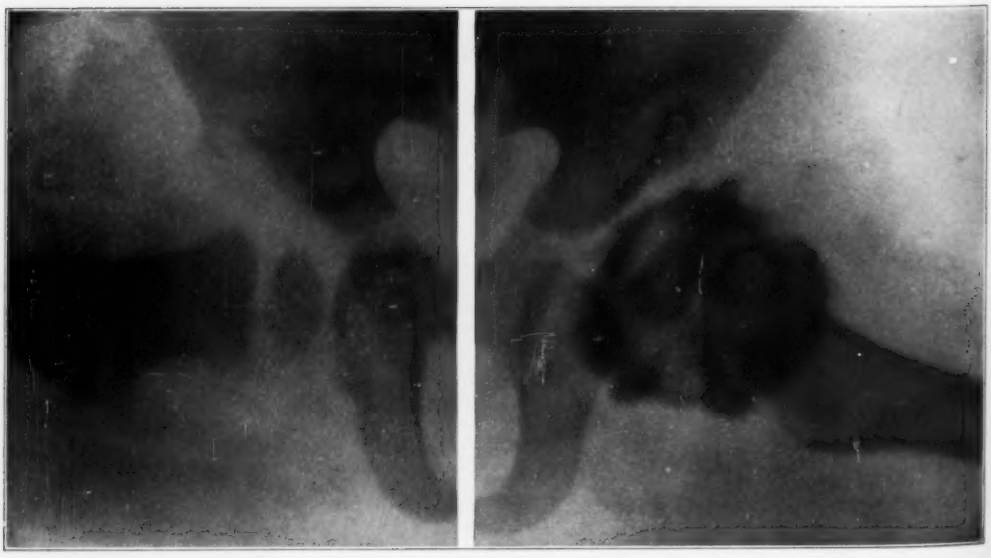

FIG. 7-B

Figs. 7-A and 7-B: Arthrograms of a girl, two years old, showing insufficient reduction of subluxation of the left hip (May 1946).

FIG. 7-C

Immediate result following open reduction by the intra-articular method, without resection of the neck (July 1946).

(*a*) *Open Reduction by the Extra-Articular Method*: For a long time I resorted to a technique based upon that of Zahradníček. This operation, which is entirely extra-articular, consists in lowering the head into the acetabulum by correcting the valgus and the anteversion through a wedge osteotomy at the base of the neck,—a wedge which has both a base distally to correct the valgus and a base posteriorly to correct the anteversion. The operation is concluded by lowering the osseous roof, which is held in place by a graft, taken from the fragments resected from the neck (Fig. 5).

This method, which I have used in fifteen cases, gave good reductions in only a few patients. In most cases there remains an abnormally wide gap between the head and the bottom of the acetabulum, the head being too big to penetrate the cavity. In these conditions a good articular congruency can only be obtained by some scraping out of the bottom of the acetabulum, especially in its lower segment. For this reason, I have now resorted to open reduction by the intra-articular approach.

(*b*) *Open Reduction by the Intra-Articular Approach*: This technique has been used by me in some thirty cases to date.

First, Ollier's transtrochanteric approach is used, which allows all necessary corrections to be made on the various elements of the articulation. After the trochanter major and the gluteus medius muscle have been raised, the capsule is cut circularly. The opening into the acetabulum is extremely large. The various moves in the operation are as follows:

1. Scoop out the acetabulum with a curette or gouge. This is done under most favorable conditions in young children, in whom the layer of cartilage covering the acetabulum is thicker.

2. Free the capsule from its adhesions to the external iliac fossa.

3. Lower the scooped roof by means of a slightly oblique osteotomy, reaching the cartilaginous covering of the acetabulum without cutting through it. The lowered roof will be held in place at the end of the operation by a graft taken either from the upper extremity of the femur, or from the wedge-shaped fragment resected from the base of the neck, when this resection has been made.

4. When accurate measurements have shown that there is a pronounced degree of valgus and anteversion, a trapezoid resection is made at the base of the neck, after

TABLE I

DIFFERENCES BETWEEN SUBLUXATION AND DISLOCATION

	Subluxation	Dislocation
Limbus and cartilaginous roof	Pushed upward against the ilium Atrophied	Pushed downward into the acetabulum Hypertrophied
Acetabulum	Early becomes oval in shape because of atrophy of roof	Normal Entrance restricted by displaced glenoidal labrum
Femoral head	Hypertrophied Enlarged transversely	Normal or atrophied
Capsule	Never interposed	Frequently interposed
Ligamentum teres	Absent	Present in 33 per cent. Often hypertrophied
Valgus	Developed early, if not primary	Developed late and secondary
Anteversion	Developed early, if not primary	Developed late and secondary

Zahradníček's method. The two osseous fragments of the neck are then fastened with a nail, triangular in section.

5. At the end of the operation, the capsule is sutured only at its upper part, after all the extruding parts have been resected.

The limb is then put in plaster, in a position of slight abduction and internal rotation, and is held in this position for a month. At the end of the month, exercises are begun. The patient gets up in the third month. During convalescence, great care must be taken that the articulation retains a correct position. If there is a tendency to flexion, adduction, or external rotation, this will be corrected by usual orthopaedic methods.

The mortality resulting from this rather delicate operation has been nil. I have to date performed consecutively more than 250 open operations in cases of subluxation or luxation of the hip (the technique being analogous) without losing a single patient. Such good results are due, not only to the perfection of the technique, but also to the use of a preventive treatment for postoperative shock (intravenous perfusion of physiological saline with the addition of adrenalin as needed).

No postoperative osteochondritis has been observed. After trapezoidal resection of the neck, the seemingly threatening dan-

FIG. 8-A

FIG. 8-B

Fig. 8-A: Roentgenogram (Feb. 1937) of a girl, twelve years old, showing subluxation of the left hip.
Fig. 8-B: Apr. 1937. Following reduction by the intra-articular method (with resection of the neck).

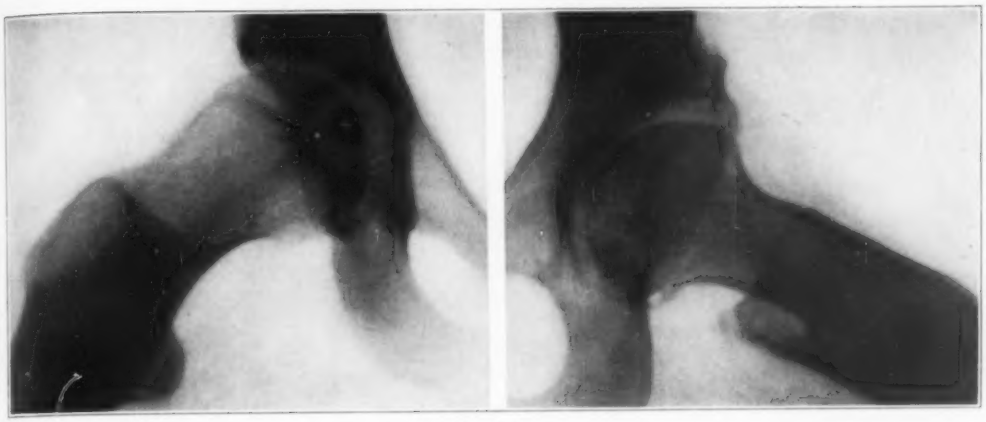

FIG. 8-C

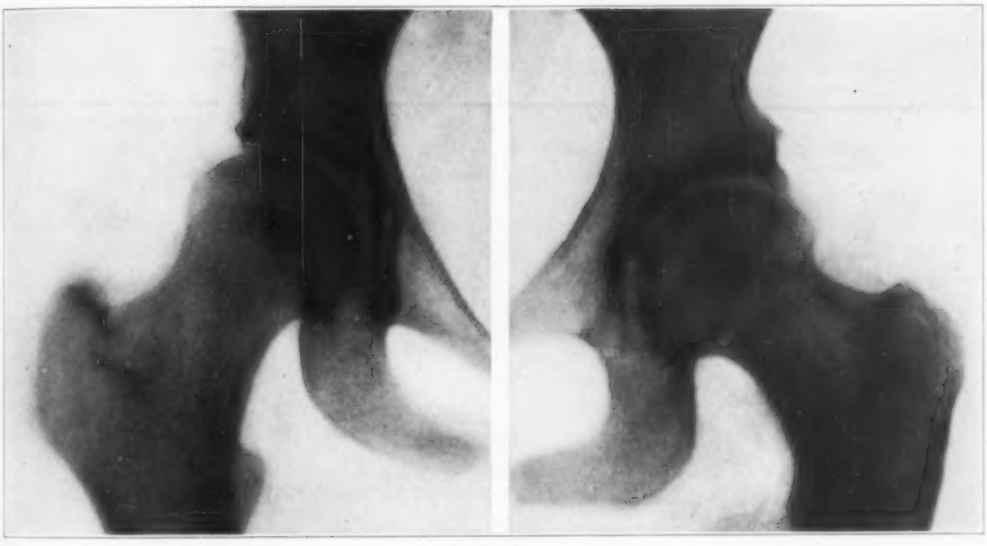

FIG. 8-D

Good result eight years after the operation.

ger of resorption of the head through insufficient vascularization is actually very small. I have observed only one case, in one of my first patients, when I had probably been too careless with the musculocapsular flap of the lower segment of the neck, through which a good circulation in the femoral head is easily maintained.

The anatomical results are always very satisfactory and well-nigh perfect, when all operative details have been followed. The functional results, especially the mobility of the joint, depend mostly upon the age of the patient and upon the extent to which the acetabulum has had to be scooped out. In young subjects the articulation always recovers normal, or near normal, mobility. In older patients some stiffness is observed, which, as a rule, is compatible with very satisfactory joint function. This stiffness tends to diminish with time.

At present, it is too early to say anything about late results, since my earliest operation by the intra-articular method was performed only eight years ago.

Figures 8-C and 8-D show the result of my earliest operation by this method, which is really encouraging. Results of this order are, of course, obtained more easily when operating upon young subjects, in whom resection of the base of the neck is unnecessary.

We have every reason to hope that this logical reconstructive surgery, which restores

the congruity of the articulation, will safeguard these patients against deforming arthritis. Only the future can show whether the late results will fulfill that hope.

NOTE: The illustrations have been reproduced from the author's book, entitled *"Luxations et Subluxations Congénitales de la Hanche. Leur Traitement Basé sur l'Arthrographie"*, by permission of Gaston Doin et Cie.

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THE USE OF PARALLEL GRAFTS AND OF TWO-STAGE AND THREE-STAGE INTERLOCKING GRAFTS IN THE TREATMENT OF IDIOPATHIC SCOLIOSIS

End Results in Forty-One Cases

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In the past several years, the few articles published on the subject of severe or progressive idiopathic scoliosis have consisted chiefly of excellent reviews on the treatment of this condition[3,7,8,10]. The use of the Risser turnbuckle jacket to correct the predetermined primary curve, followed by spine fusion to maintain this correction, is the method advocated chiefly.

LeMesurier and Blount and Schmidt have sought to simplify the method of obtaining correction. LeMesurier has proposed a hammock suspension cast, which is said to secure as much correction as the hinged jacket in an easier and less time-consuming manner. Blount and Schmidt have devised a corrective brace which they believe supersedes the tedious and prolonged method of Risser.

Whatever the merits of these two methods may be, neither meets the greatest need. An analysis of the report of the Research Committee of The American Orthopaedic Association[9] suggests definitely that the chief problem is not an easier method of obtaining correction, since the average correction attained was 61 per cent.; but the need is rather for a better means of maintaining the correction, since the average correction at end result was only 25 per cent. An even greater need is for a method which will eliminate or minimize the high incidence of pseudarthrosis, which amounted to 28 per cent. in the Committee's report, and of delayed fusion. In cases of delayed fusion, the fusion occurs after most or all of the correction and balance have been lost. Recorrection can only be obtained after an osteotomy of the spine has been performed through the fused area.

By a modification in the technique of using tibial grafts in spine fusion, developed by the senior author, apparently significant improvement has been achieved in both of these directions, when the present results are compared with those previously obtained by the Hibbs method of spine fusion, as well as with those reported by the Research Committee.

PROCEDURE

The technique used in fusing the corrected portions of the spine was developed gradually in a series of steps. Early, with short curves which included only six or seven vertebrae, parallel tibial grafts were placed on either side of the spinous processes in two stages (Fig. 1). With this type of procedure it was hoped that, if fusion was not secured in a greater percentage of cases, it would at least be easier to detect a fractured graft than to demonstrate pseudarthrosis in a spine fusion, done by the use of multiple chip grafts. As a matter of fact, the first hope was realized more than the second; for, while the incidence of fractured grafts was very low, the grafts often fused so completely and rapidly with the host bone that they very early lost their identity.

In longer curves, where it was not possible to apply the tibial grafts in a parallel manner, they were spread apart to include the entire curve and were interlocked in the center, thus giving the maximum support at the point of greatest stress. These were termed two-stage interlocking grafts (Fig. 2). Finally, in long curves, it was decided to use three-stage interlocking grafts. This was accomplished by putting the first graft on one side of the spinous processes, the second graft on the other side, and the third graft on the original side (Fig. 3). Each graft overlapped a minimum of two vertebrae.

In each stage, the procedure consists in exposing only one side of the spinous processes and laminae. A bed for the tibial graft is made by turning down bone chips from the

FIG. 1

FIG. 2

Fig. 1: Parallel grafts.
Fig. 2: Two-stage interlocking grafts. (The corrective jacket has just been removed and decompensation of the lumbar curve has not yet occurred.)

FIG. 3

FIG. 4

Fig. 3: Three-stage interlocking grafts (in a double primary curve).
Fig. 4: Fracture of graft on left side, just above tenth rib.

spinous processes and laminae without attacking the lateral articulations. Then, with the graft in place, the spinous processes are undercut and, by the use of several Lembert sutures through the muscle and periosteum, the spinous processes are inverted to come into close contact with the graft.

In the more severe curves, particularly in patients with increased anteroposterior curves, it is necessary to secure curved tibial grafts. However, since the segments are relatively short in the two-stage and three-stage interlocking procedures, this is almost always possible to accomplish. In the few cases in which it is not possible, equal difficulty would be encountered in doing the classical Hibbs procedure, since the convex side of the vertebral curve is almost inaccessible, as the laminae and facets are rotated, twisted, and overhung by the spinous processes and the distorted convexity of the ribs. The procedure of Henry and Geist is usually followed in these severe cases.

The necessity for two incisions, one of which results in a scar on the leg—especially for a condition in which the chief purpose of correction is cosmetic—is admittedly a distinct disadvantage. Nevertheless, the advantages of this method of spine fusion outweigh the disadvantages. The procedure produces less shock, because only one side is exposed and dissection need not be carried out to the lateral articulations, which are often inaccessible in severe curves; the incidence of pseudarthrosis is greatly decreased; and the percentage of correction maintained as an end result is significantly increased.

COMPLICATIONS

Mortality: In this series of forty-one cases of idiopathic scoliosis treated by jacket correction and spine fusion (which included ninety-one operative procedures), there was a mortality rate of 0 per cent.

Aseptic Necrosis of Graft: In one case (2.4 per cent.), after primary healing, a drain-

FIG. 5-A

FIG. 5-B

Fig. 5-A: Film, taken before treatment, shows curve from eleventh thoracic to third lumbar vertebra. This was selected as the primary curve, because it is the middle curve, it is the greatest curve, and the vertebrae are most rotated.

Fig. 5-B: Shows end result. In spite of correction of the curve from 37 degrees to 8 degrees, decompensation of the thoracic curve did not occur, indicating that this is also a primary curve.

ing sinus developed which persisted, and roentgenograms showed aseptic necrosis of a portion of the graft. This fragment was removed, and, because of the interlocking technique used, with ample overlap of the grafts, no particular loss of correction occurred.

Fracture of Graft: In only three cases (7.3 per cent.) did fracture of a graft occur. In one patient a fracture developed at the end of the middle graft in a three-stage interlocking procedure, apparently before the third stage was done. Since there was sufficient overlap of the other graft, no undue loss of correction developed and no further surgery was necessary. In a second patient the fracture was found early, before there was much loss of correction, and a repair of the pseudarthrosis was carried out. The third patient has recently been re-admitted for surgery. In each instance the fractured graft was easily demonstrated by roentgenogram (Fig. 4). This represents an advantage of this operative procedure over the Hibbs technique, in which it is very difficult to make an early diagnosis of pseudarthrosis.

Fracture of Tibia: One patient fractured the tibia from which a graft had been removed.

Pressure Areas: Mild pressure areas developed in three cases.

Partial Brachial Palsy (Transient): Five patients (12.2 per cent.) had mild, transient, partial brachial palsy.

Wound Infection: There was one case of mild infection of a leg wound.

Double Primary Curves: Although not truly a complication, the double primary curve may well be considered here. If unrecognized, it can cause an unsatisfactory end result, even though the chosen primary curve has been well corrected and successfully fused (Figs. 5-A, 5-B, and 6).

The criteria used in selecting the primary curve[10] are generally known, and have proved of great value if there is a single primary curve. Unfortunately these rules do not apply to the double primary curve, and no certain method for detecting such a curve is at present available. Von Lackum's observations in this regard are of distinct help.

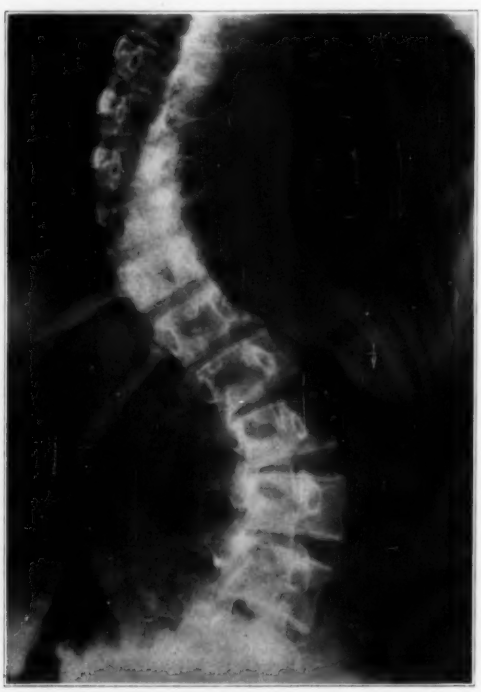

FIG. 6

Roentgenogram of a double primary curve. It was so labeled because the lumbar curve did not straighten in the tilt test, the thoracic curve was clinically rigid, and the vertebrae showed wedging and rotation.

If a patient with a double primary curve is discovered, several methods of treatment have been advocated:

1. The most deforming curve is corrected and, if the end result is unsatisfactory, the second curve is corrected and fused.

2. The thoracic curve is corrected, and then apparent balance of the involved pelvis and the prominent hip is induced through the use of a heel lift[5]. This is not too happy a solution, since the patient, after undergoing rather strenuous treatment, must still depend upon mechanical aid.

3. The thoracic curve is corrected as much as possible, then the lumbar curve is corrected to an equal amount, and fusion of the entire area is carried out.

This last procedure proved most successful in the five cases of double primary curves in this series. The correction with a Risser jacket is done by using two sets of hinges and two turnbuckles. First the thoracic curve is corrected; then the lumbar curve is straightened

TABLE I

AVERAGE PERCENTAGE OF CORRECTION FOR EACH CURVE

Type of Curve	Report of Research Committee			Present Series		
	No. of Cases	Correction after Operation (*Per Cent.*)	End Result (*Per Cent.*)	No. of Cases	Correction after Operation (*Per Cent.*)	End Result (*Per Cent.*)
Mild (0 to 24 degrees)	10	71	33	1	94.0	80.0
Moderate (25 to 44 degrees)	51	72	25	11	71.5	49.6
Severe (45 degrees and over)	88	53	24	24	57.3	40.0
	149	61	25	36	62.6	43.1

equally. This method obviates the necessity of applying two corrective jackets, and materially shortens the time of treatment. Finally, the entire area is fused by using the necessary number of interlocking tibial grafts (Fig. 3).

END RESULTS

Table I shows that the end results in this series of cases of idiopathic scoliosis are substantially improved over the results indicated by the Research Committee. These patients have, for the most part, been followed for from two to five years, and the shortest follow-up period is well over a year after operation. Successive roentgenograms indicate that no gradual loss of correction is occurring.

Since corrective treatment is undertaken with the idea of eliminating or decreasing the degree of curvature, the success or failure of the method must be judged on this basis. However, the cosmetic end result is more directly related to the restoration of body balance than it is to the number of degrees of correction (Figs. 7-A and 7-B).

The preoperative and end-result photographs of approximately average cases (mild, moderate, and severe) are shown in Figures 8-A, 8-B, 9-A, 9-B, 10-A, and 10-B.

An analysis of the end results in this series indicates that over half the cases treated are classified as severe. A definite cause of this is the practice of treating early cases of idiopathic scoliosis with casts and braces for long periods of time, without adequate roentgenographic follow-up. Forty-nine per cent. of these cases were so treated early in the disease, and in all progression continued.

In treating scoliosis with the types of braces commonly used, one attempts to correct the spine indirectly through pressure on the thorax, which is an elastic structure; and thus a great deal of the force exerted will be spent merely on increasing the angulation of the ribs or changing the relations at their costovertebral joints[1]. At best, the method is of questionable value. If it is used at all, it certainly should not be persisted in to the point of allowing a mild curve to progress to the moderate or severe stage.

The most satisfactory early approach to the problem is a careful check on the progress of the curve by direct measurements of roentgenograms taken every three months. Postural exercises are often used during these intervals. Curves that show progression should be considered for correction by the Risser jacket and spine fusion, always remembering that some curves will progress, but finally become arrested before they reach a degree which is clinically important.

Although it is the authors' chief purpose to present operative procedures and end results in the treatment of idiopathic scoliosis, a brief discussion regarding the methods of obtaining correction seems within the scope of this paper.

Although the Risser jacket was used in all but two cases to obtain correction of the curvature, in six cases the hammock suspension method of LeMesurier was tried. Four cases were classed as severe, one as moderate, and one as having a mild curve.

Of the severe curves, a 76-degree curve was corrected in the hammock to 70 degrees

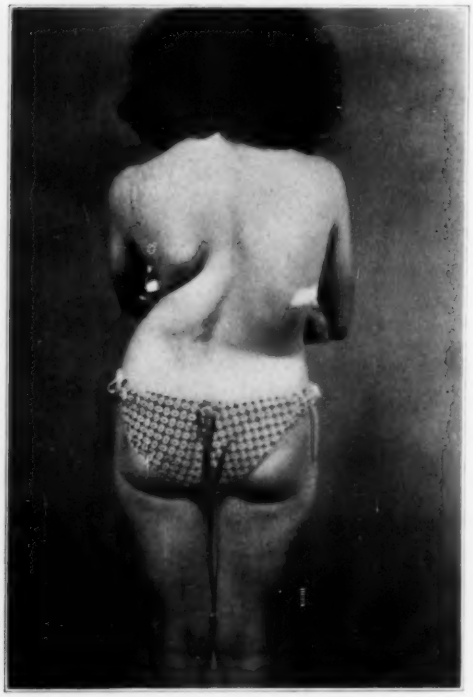

FIG. 7-A

FIG. 7-B

Fig. 7-A: This patient had a severe curve,—58 degrees before correction.
Fig. 7-B: At end result, only 13 degrees of correction was retained. The statistical result is poor, yet the clinical result is very acceptable.

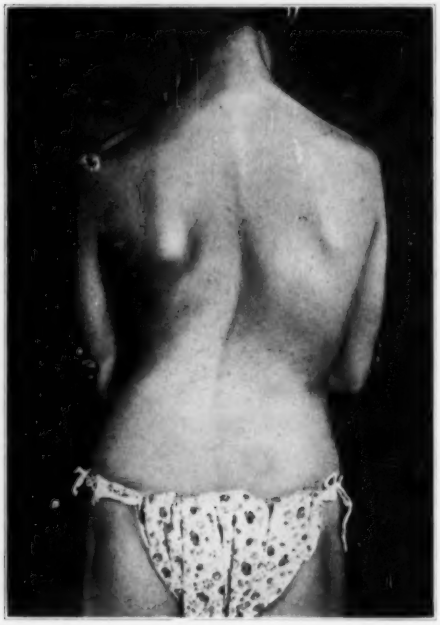

FIG. 8-A

FIG. 8-B

Photographs of a mild curve before correction and at end result.

FIG. 9-A

FIG. 9-B

Patient with a moderate curve, shown before correction and at end result.

FIG. 10-A

FIG. 10-B

Photographs of a severe curve, shown before correction and at end result.

and in the Risser jacket to 32 degrees; a 53-degree curve was corrected to 39 degrees in the hammock and to 20 degrees in the jacket; a 63-degree curve became 48 degrees by suspension, but decreased to 34 degrees in the turnbuckle jacket; and an 82-degree curve was decreased in the hammock to 55 degrees and in the Risser jacket to 47 degrees. All of these patients were suspended in the fish net from one week to one month before being placed in the Risser jacket. However, the mild curve of 17 degrees and the moderate curve

of 26 degrees were corrected down to 2 degrees by this method, and these patients were placed in plaster jackets while suspended in the fish net.

SUMMARY

Significant improvement in the amount of correction retained and a substantial decrease in the incidence of pseudarthrosis have been achieved in forty-one cases of idiopathic scoliosis. These patients were treated by a modified technique of using tibial grafts in spine fusion, after obtaining maximum correction (by the Risser type of plaster jacket in thirty-nine cases and by the hammock suspension method in two cases).

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VITAMIN-A DEFICIENCY AND EXCESS IN RELATION TO SKELETAL GROWTH * †

BY S. BURT WOLBACH, M.D., BOSTON, MASSACHUSETTS

Bone is a peculiarly favorable tissue for the study of growth, because growth is not simply increase in size; it involves alterations of form. Bone, we know, undergoes continuous remodelling, rapidly during growth, slowly thereafter; and, because of this and similar responses common in pathological conditions, it is to be regarded as an exceedingly labile tissue. Fortunately, remodelling activities of bone leave a trail which can be read, a trail blazed by etched haversian systems, interrupted lamellae, and denser calcium deposits in the so-called cement lines which separate old from newly formed bone. The agents that cause resorption of bone are probably multiple, one of which will be considered in particular in this study. The information obtained from study of bone pathology and from a few connective-tissue behaviors, observable during cyclic physiological processes, leads to the conviction that all our collagenous structures are as labile as bone, subject to constant processes of resorption and new formation. Failure of such adjustments may be regarded as one consequence of age; such failure, indeed, may be the result of irreversible collagenous compounds, slowly accumulated and not amenable to the processes involved in maintenance.

All vitamin deficiencies that culminate in severe malnutrition (and most of them do) arrest growth of bone. Malnutrition caused by inadequate amounts of a perfect diet also arrests bone growth. In every instance where bone growth has ceased because of general inanition, a sturdy bone with normal contours is the result.

There are three vitamin deficiencies that affect bone growth directly. These are A, C, and D. The operations of A and D in bone growth specifically involve epiphyseal-cartilage-cell activities. In vitamin-C deficiency in growing animals, bone suffers in common with all collagenous structures.

It seems self-evident that lack of a vitamin causes suppression of the biochemical systems in which the vitamin is operative.

Vitamin A is unique, because it is the only vitamin which, as will be shown, when given in quantities greatly in excess of normal requirements, actually accelerates some normal growth sequences.

I have proof that the degree of acceleration of growth sequences bears quantitative relationships to the amount of vitamin A administered, and can be pushed to dramatic and disastrous consequences upon bones. In considering the effects of excessive introduction of vitamin A, we may speculate either in terms of increased reaction velocities or of increase in numbers of biochemical systems.

Vitamin A in deficiency and excess in relation to skeletal growth is my subject. I shall outline some of the premises which have led me to the conclusion that Vitamin A is specifically involved in mammalian skeletal growth. Both a deficiency and an excess of vitamin A affect profoundly the growth of bone. The deficiency suppresses, the excess accelerates epiphyseal-cartilage-cell sequences. I shall outline the consequences of such changes in epiphyseal-cartilage sequences upon the remodelling of bone which is an essential feature of bone growth. I shall present evidence which indicates that remodelling sequences attending growth of bone are induced by a factor elaborated in the maturation of epiphyseal-cartilage cells.

* Twenty-Second Ludvig Hektoen Lecture of the Frank Billings Foundation delivered before the Institute of Medicine of Chicago, March 22, 1946.

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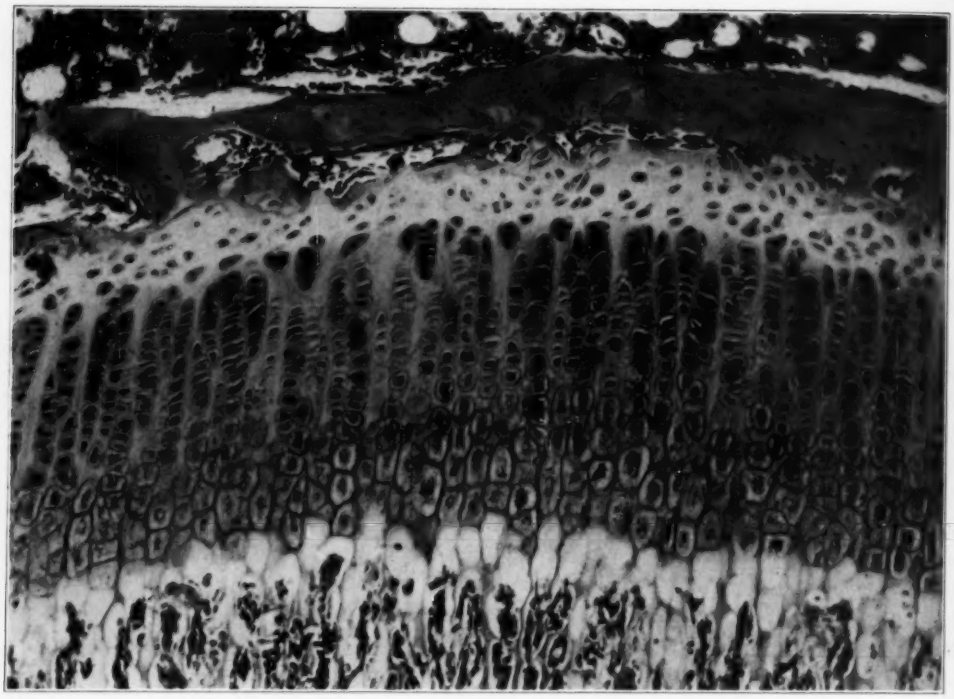

FIG. 1

Proximal epiphyseal cartilage of tibia of guinea pig, illustrating cytomorphosis of cartilage cells and growth of capillaries into spaces left by death of cells.

The substance of this lecture should be presented against a background, provided by an outline of normal bone growth and the effects thereon of vitamins C and D deficiencies.

Increase in size of most bones during growth of the skeleton—all those which are first laid down as hyaline cartilage in the embryo, including all of the long bones, the vertebrae, and bones of the base of the skull—is by endochondral bone formation for which purpose, until growth is completed, cartilage persists at epidiaphyseal junctions. Growth of a long bone, for example, is accomplished by continuous proliferation of cartilage cells, arranged in columns on the epiphyseal side, and concurrent degeneration of matured cartilage cells on the diaphyseal side. The spaces which result from the degeneration and disappearance of the cartilage cells at the diaphyseal ends of the columns are penetrated by capillaries, accompanied by osteoblasts, which are responsible for the deposition of bone matrix upon the exposed cartilage matrix. Multiplication, increase in size, maturation, and death of cartilage cells (cytomorphosis)[10] are, therefore, necessary sequences in normal skeletal growth[13] (Fig. 1).

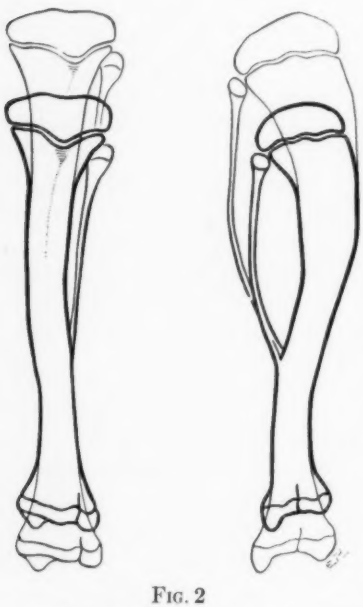

FIG. 2

Tibia and fibula of rat. Diagram to illustrate remodelling during growth, viewed from anterior aspect and in profile.

However, growth of bone is not a one-way process; preservation of conformation demands continuous remodelling, involving resorption of bone where reduction of size is necessary and new deposition of bone from endosteal and periosteal sources to preserve continuity of structure (Fig. 2). The deposition of bone by cells

of endosteum and periosteum we refer to as appositional bone formation. Some bone contours, especially those where ligaments, fasciae, tendons, muscles, and dura are attached, are built up by appositional bone formation. The growth of the few bones which developed directly in connective tissue—membraneous bone formation—including the bones of the calvarium, sides of the skull, and most of the facial bones, is by appositional bone growth at the sutures and from periosteum and endosteum. Such bones also undergo remodelling during growth, but I have good reasons for the conviction that the pattern is set by the growth of those bones of the skull which are of cartilaginous origin. In general, endochondral bone formation sets the pace and the pattern of skeletal growth. In the microscopic study of bone, one can usually get a clear picture of what is happening and, indeed, of what has happened, because the remodelling process leaves distinctive features. Regions of resorption are usually indicated by the presence of osteoclasts and breaks in continuity of lamellae. Regions of appositional bone formation can be identified easily by the presence of rows of osteoblasts and non-calcified matrix or osteoid. Where remodelling has occurred, boundaries between resorbed bone and bone subsequently deposited are indicated by cement lines, as well as by discontinuities of lamellae (Fig. 3).

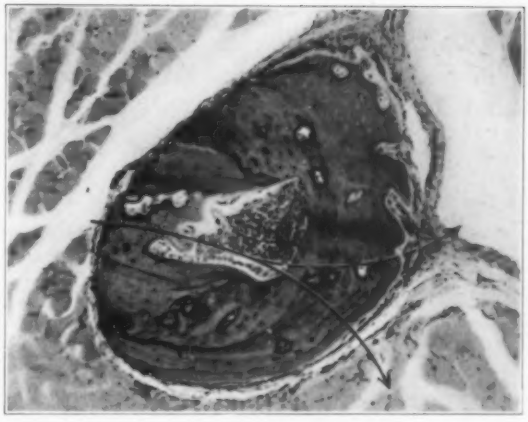

FIG. 3

Cross section of fibula of normal rat to illustrate the pattern of remodelling in consequence of changes in its relation to the tibia, as indicated in Fig. 2. The arrows indicate the outward and posterior swing of this bone.

VITAMIN-C DEFICIENCY

We know of no untoward effect resulting from administration of excessive amounts of vitamin C. Man, monkeys, and the guinea pig are the only animals now known which are unable to synthesize vitamin C. The lesions resulting from vitamin-C deficiency in the young of these animals are the consequence of the failure of formation of intercellular materials. Bone formation, endochondral and appositional, is prevented because of the failure of matrix formation, specifically a failure in the synthesis of the protein or group of closely related proteins,—collagen. All evidence indicates that the physiology of calcification is not involved[15,16].

This failure of bone-matrix formation explains perfectly the gross pathology of vitamin-C deficiency or scurvy in infants. Cartilage sequences in endochondral bone formation continue, but cells accompanying the ingrowing blood vessel can deposit no matrix and hence discontinuity of epiphysis and diaphysis—infraction—takes place. Periosteal cells concerned in appositional bone growth through failure of matrix deposition permit the fibrous periosteum to pull away from bone. Not only is there failure to form collagenous matrices, but there is good reason for believing that resorption of matrices of bone and connective tissue takes place. Remodelling of non-calcified supporting tissues leaves no trail.

The administration of vitamin C to totally deficient guinea pigs is followed by prompt deposition of collagen and formation of the matrices of connective tissue, bone, and teeth. Careful studies by Boyle, Bessey, and Howe have shown that the amount of dentine matrix (predentine) formed is quantitatively related to the amount of vitamin C administered. The relation of vitamin C to growth and maintenance of bone resides solely in its effect upon the formation and maintenance of the organic matrix.

VITAMIN-D DEFICIENCY

Normal growth of bone in the human is dependent upon the action of vitamin D in maintaining efficient concentrations of calcium and phosphorus ions in the blood plasma. In the rat, normal bone growth in the absence of vitamin D continues, unless the diet is such as to produce in the blood a "relative deficiency of calcium or phosphorus or an absolute deficiency of either or both" [13].

It is not necessary to present the pathology of rickets in detail, because this would lead into controversial fields, some of which would not exist if more attention had been given to early recovery sequences in cure of experimental rickets. Experimental rickets in rats reproduces accurately the spontaneous disease in human infants and other animals. As in all deficiencies, an understanding is to be sought in terms of retardation or suppression of normal processes, in this instance growth sequences in epiphyseal cartilage and of calcification of bone and cartilage. The reason why the pathology of human rickets is apparently so complicated is that the deficiency in any individual studied may have been intermittent or of varying degrees from slight retardation to almost complete suppression. Endochondral growth of bone is dependent upon proliferation, differentiation, and degeneration, ending in death and disappearance of cartilage cells [17]. In rickets the cartilage cells fail to complete this sequence; they reach their maximum size, produce adequate amounts of matrix, but do not degenerate. For this reason, as proliferation continues, the epiphyseal cartilage accumulates. In the absence of spaces created by the disappearance of cartilage cells, there is no ingrowth of capillaries. Concurrently with the arrestment of cartilage-cell sequences, the matrix adjacent to the persistent cells fails to calcify. Absolute rickets in the sense of the complete cessation of the above sequences is of doubtful occurrence in humans and difficult of experimental achievement. The arrestment takes place irregularly across the diaphyseal face of the cartilage plate. Wherever cartilage cells degenerate, vascular ingrowth takes place, in normal direction or laterally as opportunity offers; but the bone matrix (osteoid) deposited about the capillaries does not calcify.

FIG. 4

Proximal epiphysis of tibia of rat. Showing repair of experimental rickets after seventy-two hours, and illustrating resumption of cytomorphosis.

Deposition of bone matrix (osteoid) continues to take place around capillaries of the

diaphysis adjacent to the cartilage. The accumulated osteoid and cartilage may become molded or distorted by the pressure of weight-bearing, in cartilage frequently resulting in stratification. In long-continued rickets there is disappearance of trabeculae of the diaphysis and resorption of cortical bone (osteoporosis), in response to more vital needs of the body for calcium.

The first cytological evidence of repair in the rat following vitamin-D therapy is the resumption of cytomorphosis (Fig. 4) visible in twenty-four hours through the presence of cleared or degenerated cells on the diaphyseal border of the cartilage, and accompanied by extensive penetration of capillaries within forty-eight hours, and concurrent calcification of adjacent matrix. The complicated patterns of capillary tufts in the epiphyses of rickets, which have occasioned much discussion and speculation by various authors [11], are satisfactorily explained by the occurrence of irregularly distributed groups of cartilage cells which have completed their sequences, a result of varying degrees of intermittency of the deficiency across the face of the epiphyseal plate. Thus, opportunities for ingrowth of capillaries in lateral and even diaphyseal directions can exist. Such capillary patterns may be seen in the early repair stages of advanced experimental rickets.

Calcification of cartilage matrix and of osteoid first takes place adjacent to capillaries which have entered the spaces left by the degenerated cartilage cells, wherever this has occurred. Subsequently the calcification of accumulated osteoid progresses toward the diaphysis. Excess osteoid, which has accumulated as a result of the deficiency, is removed only after calcification by osteoclasis. Apparently there is no mechanism for the removal of non-calcified bone and cartilage matrices.

Thus in rickets, two separate processes are involved,—one the failure of epiphyseal

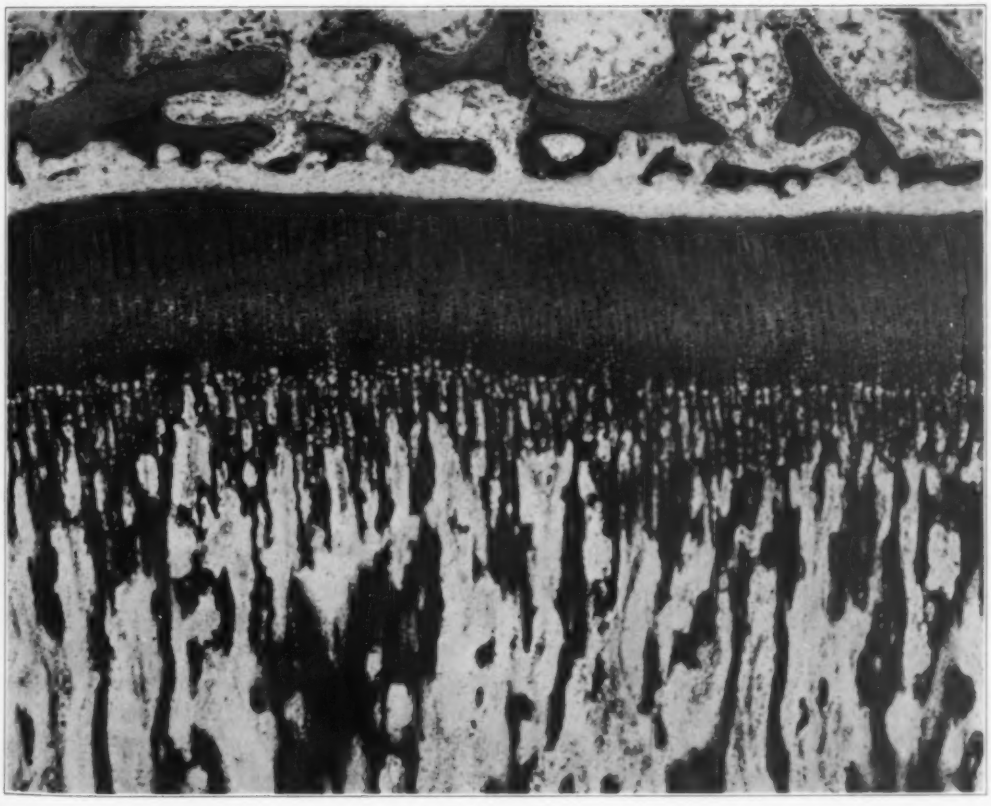

FIG. 5

Proximal epiphysis of tibia of normal litter mate, control puppy to the vitamin-A deficient puppy, the source of Fig. 6.

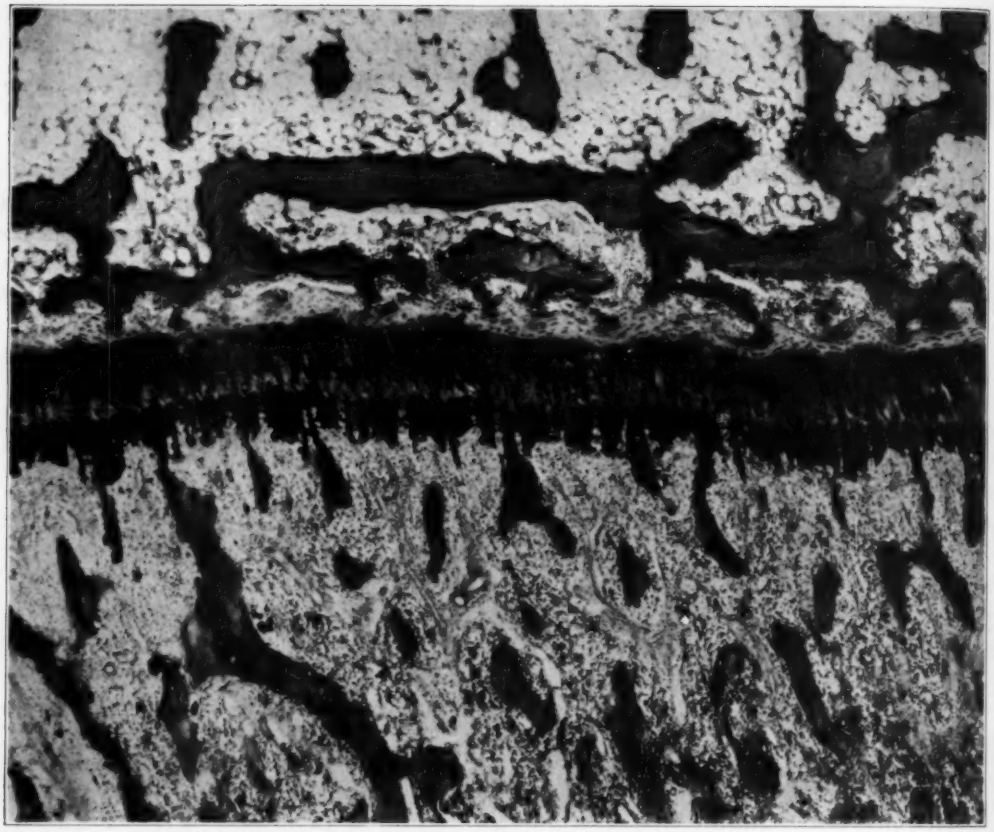

FIG. 6

Proximal epiphysis of tibia of vitamin-A deficient puppy of the same litter as source of Fig. 5.

cartilage cells to complete the sequences of proliferation, maturation, and degeneration, the other the failure of matrices to calcify.

A comment made some years ago in regard to experimental rickets and which applies also to osteomalacia is perhaps worth repeating: "The degree or severity of rickets may be estimated and recorded on the basis of the prominence of anatomical changes demonstrable by roentgenograms or histological study. Obviously two factors enter into the production of the pathological picture, the duration of the deficiency and the degree of the deficiency as measured by calcium and phosphorus blood concentrations and ratios. Either factor can be made the variable and thus the time factor can be calibrated against the chemical factor — in so far as increased width of epiphyseal cartilage and accumulation of osteoid are concerned" [17].

HYPERVITAMINOSIS D

The administration of excessive amounts of vitamin D (activated ergosterol) produces untoward effects in bone and soft tissues, resulting from a great increase in serum calcium [1]. The physiological role of vitamin D is still generally regarded as one operative in the process of absorption of calcium from the intestines. The fact that, if the alimentary source of calcium is not adequate, calcium is obtained through resorption of bone [6,12] suggests that a more important role is to be found in the establishment of equilibrium conditions in the blood plasma. The stoichiometry of blood calcium seems to have quantitative relations with the amount of vitamin D introduced. The maintenance of high calcium levels in rats through its use results in short bones with non-calcified trabeculae of unusual thickness and number at the epiphyseal ends and resorption of cortical bone with non-

calcified matrix deposition of periosteal and endosteal origin. These changes have led one group of authors [5,6] to regard the effect as rachitic in type, in spite of the fact that epiphyseal-cartilage-cell sequences are not suppressed. The persistence of essentially normal cartilage-cell sequences under conditions which cause resorption of bone and prevent calcification of newly deposited bone matrix suggests either that the cartilage cells participate in alterations of equilibrium conditions affecting calcium, or that they do not require calcium but do require vitamin D for the sequence of growth, maturation, and degeneration. A single massive dose of activated ergosterol [4] causes a great rise of serum calcium, followed by deposition of calcium salts in many soft tissues concurrently with the fall in serum calcium.

VITAMIN-A DEFICIENCY

In vitamin-A deficiency, all epiphyseal-cartilage sequences cease (Figs. 5, 6 and 7). Cells cease to divide, and cells which have reached almost mature size undergo no further change. Only those cells which have progressed to the vesicular stage continue to change and finally disappear, permitting vascular penetration. The cartilage trabeculae calcified before the deficiency was established are resorbed by osteoclastic action. The matrix of the now atrophic epiphyseal cartilage becomes densely calcified for a depth of a few cells on the diaphyseal border, and eventually a thin bony plate is formed across the face of the epiphyseal disc. The resulting appearance is similar to that produced by inanition from inadequate amounts of a perfect diet, or by inanition resulting from other vitamin deficiencies not specifically related to bone growth,—such as riboflavin and pyridoxine. However, in inanition from these various causes, the growth of all tissues is correspondingly retarded; whereas, in vitamin-A deficiency, endochondral bone growth ceases before the growth rate of the animal as a whole is seriously affected (Figs. 8 and 9). It is this disproportionate growth of the skeleton and soft tissues which is responsible for early disastrous consequences in the young of rats, dogs, guinea pigs, and chicks, because the cranial cavity and the spinal canal become too small for the central nervous

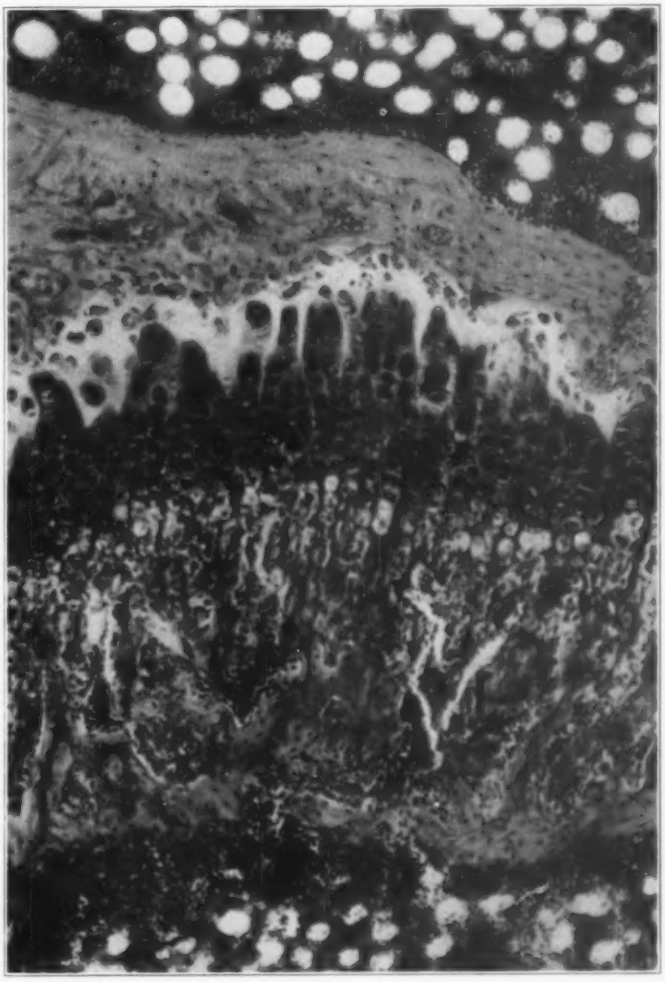

FIG. 7

Distal epiphysis of femur of rat, to illustrate the return of normal cartilage sequences and bone growth, following therapy of severe vitamin-A deficiency. Note the plate of bone on the diaphyseal side.

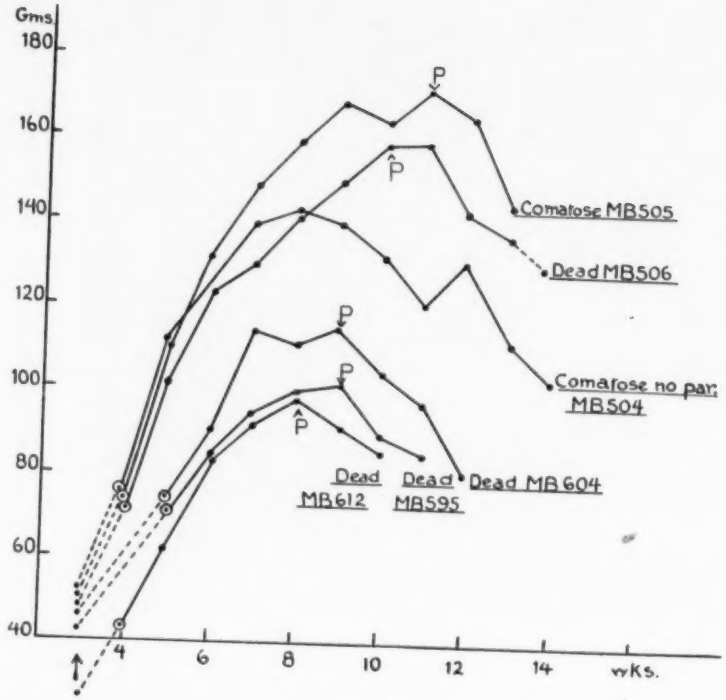

FIG. 8

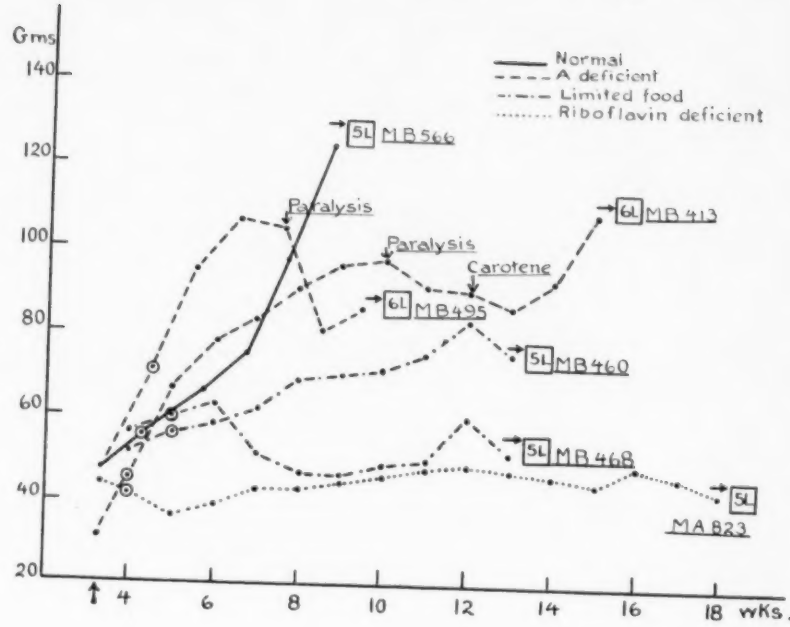

FIG. 9

Fig. 8: Weight curves of weanling rats on vitamin-A-deficient diet. To illustrate onset of paralysis, indicated by *P*, in relation to weight gain.

Fig. 9: Weight curves of two vitamin-A-deficient rats with onset of paralysis indicated. Two weight curves of rats with inanition produced by inadequate amounts of a complete diet, one weight curve of a greatly stunted rat on a riboflavin-deficient diet, and one curve of a normal rat. The rectangles at ends of the curves represent lumbar vertebrae. The arrows indicate levels of the conus terminalis. Notice that the conus of the vitamin-A-deficient rats is situated more than the length of one lumbar vertebra caudad to the normal and inanition controls (See Fig. 10). All six rats were three weeks old when placed on the experimental diets.

system which continues to grow at approximately normal rate until general inanition supervenes. The result of the disproportionate growth of bone and central nervous system is overcrowding of spinal canal and cranial cavity and paralysis, resulting from mechanical pressure upon brain, spinal cord, and nerve roots. The consequences are exhibited by multiple herniations of the cerebrum and cerebellum into arachnoidal villi, dislocation of the brain as a whole toward the foramen magnum, and buckling and herniation of nerve roots (rat and guinea pig) into intervertebral foramina and into the bodies of vertebrae. The latter is the result of atrophy of bone, caused by the pressure of buckled nerve-root trunks (Figs. 10 and 11).

Species differences in anatomy and growth patterns naturally affect the details resulting from the disproportionate growth. While identical in rat and guinea pig, there are as yet incompletely solved differences in the dog to which reference will be made later. In the chick, there are no herniations of nerve roots, because the spinal cord, unlike in most mammals, fills the entire spinal canal, and the roots leave the spinal cord through foramina at the levels of their origins in the spinal cord. However, overcrowding of the central nervous system is strikingly shown by distortion of the brain with voluminous herniations into arachnoidal villi and marked distortion of the spinal cord, shown by the cross ribbing in the lumbosacral region, which is produced by the bulging of the spinal cord into the ribbed spinal canal of this region.

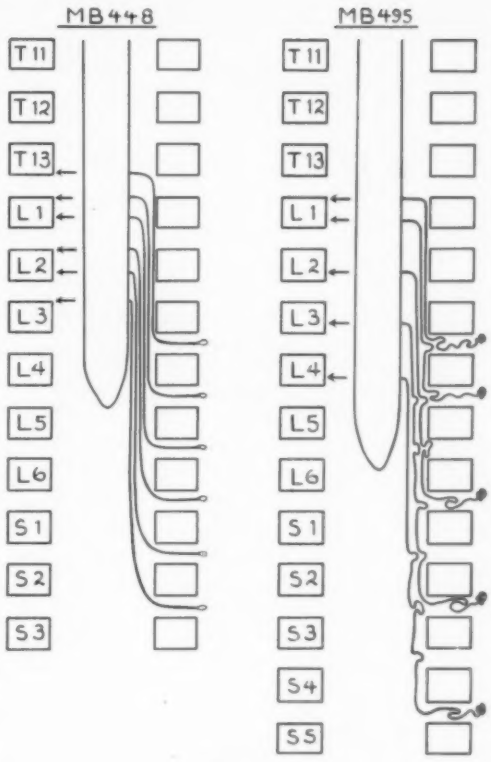

FIG. 10

Diagrams. *MB 448* illustrates the position of the conus terminalis in normal rats. The arrows indicate levels of origin from the spinal cord of a few nerve roots in relation to their foramina of exit.

MB 495 illustrates the position of the conus terminalis in a vitamin-A-deficient rat. (See Fig. 9 for weight curve.) Also, a few nerve roots are shown for sites of herniations and for levels of origins in relation to foramina of exit. This diagram is a partial record of the dissection of rat MB 495. Notice several nerve roots with multiple herniations and the low position of the conus terminalis which has resulted from the continued growth of the central nervous system after growth of the skeleton was retarded.

Cessation of endochondral growth is not the whole story of the effect of vitamin-A deficiency upon young bones; remodelling ceases and appositional bone formation continues at different rates in locations which vary with the species, but which are in conformity with each normal growth pattern.

The long bones of vitamin-A-deficient animals are shorter and thicker than those of normal animals. The shortness of the long bones is the result of failure of endochondral bone growth. The increased thickness is produced by continued appositional bone growth and, near the ends of bones, by retardation of remodelling sequences in the final period of growth before complete deficiency is established. Mellanby has paid particular attention to apparently excessive periosteal bone formation in relation to the bony labyrinth of the ear, other bones of the skull, and cervical vertebrae. Wolbach and Bessey in their 1941 paper confirmed Mellanby's findings in regard to the changes in the petrous bones of dogs, and recorded similar findings in A-deficient rats and guinea pigs. In none of Mellanby's publications [7,8,9] is mention made of epiphyseal cartilages and endochondral (replacement) bone growth.

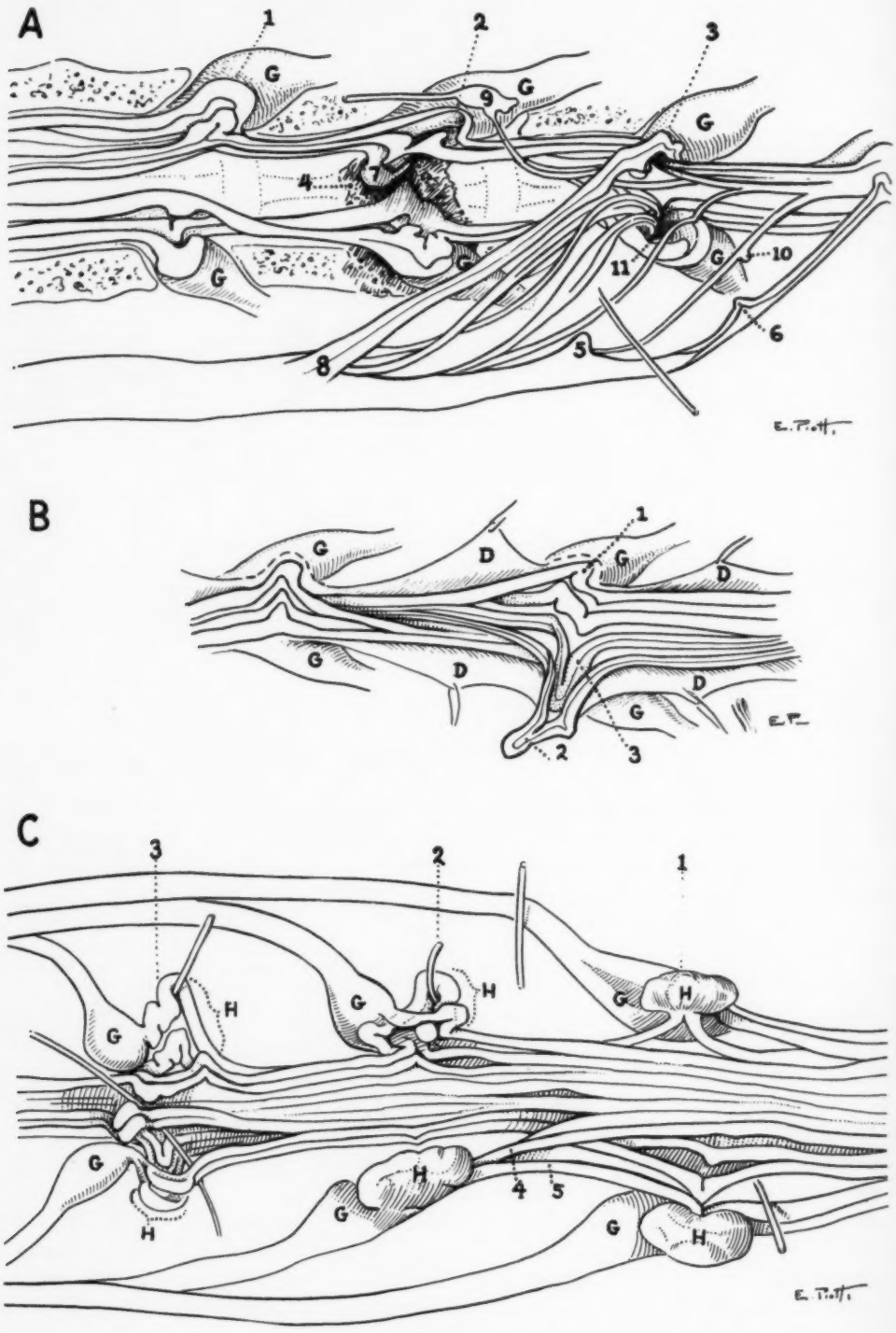

FIG. 11

A. Vitamin-A-deficient rat, ten weeks old, which had shown severe paralysis for nine days; dorsal dissection, exposing third, fourth, and fifth lumbar ganglia, indicated by *1*, *2*, and *3*, respectively.

4: Indicates where the body of the fourth lumbar vertebra has been dissected away, exposing coiled nerve roots which have pitted the bone.

5: Herniation of the spinal cord, which was extruded into the right fourth lumbar ganglion.

6: Herniation of nerve roots removed from the fifth lumbar ganglion.

7: Ventral root of the fourth lumbar ganglion, lying in a pit in the body of the fourth lumbar vertebra.

FIG. 11 (*continued*)

8: Dorsal roots to the sixth lumbar and first sacral ganglia from their origin to herniation into the fifth lumbar ganglion.
9: Herniation of a coiled and swollen unidentified nerve root, removed from the invaginated fourth lumbar ganglion.
10: Small excrescence on the ventral nerve root removed from a fifth lumbar ganglion.
11: A deep pit in the fifth lumbar vertebra with entering and emerging nerve roots.
G: Ganglia.

B. Vitamin-A-deficient rat, eleven weeks old, which had shown severe paralysis for twenty-one days; dorsal dissection, exposing fifth and sixth lumbar ganglia with herniated nerve roots.
1: Ventral root of the first sacral ganglion, herniated into the sixth lumbar, left side.
2: Dorsal and ventral roots of the third sacral ganglion, right, removed from herniation into the sixth lumbar ganglion, right.
3: Dorsal and ventral roots to the second right sacral ganglion, dislodged from herniation into the right sixth lumbar ganglion.
G: Ganglia.
D: Dura. Its line of attachment to the ganglion is represented by dotted lines.

C. Vitamin-A-deficient rat, ten weeks old, which had shown severe paralysis for nine days; ventral dissection. The bone was dissected away from the herniation occupying pits in the bodies of the fourth, fifth, and sixth lumbar vertebrae, indicated respectively by *1*, *2*, and *3*. *H* indicates herniation; *G*, ganglia. The herniations in relation to the fourth lumbar ganglia are covered by membranes. Partial dissections have been made elsewhere to give an idea of the complexity of the coils and the irregular thickening of the herniated nerve trunks. *4* and *5* indicate dorsal and ventral roots of the left fifth lumbar ganglion entering the hernial sac.

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My own studies had convinced me, several years ago, that, after endochondral bone formation had ceased in vitamin-A-deficient animals, appositional bone formation continued, and in certain locations apparently became excessive. Restudy of bones from vitamin-A-deficient animals, in the light of information obtained from the study of growth patterns of normal bones and of bones of animals which had been given greatly excessive amounts of vitamin A, has made clear the fact that appositional bone formation in the A-deficient animals continues, until inanition supervenes, *in strict conformity to normal growth patterns, both as to situation and rate.*

Some contours of long bones are formed by active appositional (periosteal) bone formation; examples in the rat are the third trochanter of the femur, the crest of the tibia, and the interosseous crests of tibia and fibula. In such regions the rate of appositional bone formation is much greater than elsewhere. Serial sections of the petrous bones of normal rats and dogs have shown that activity of appositional bone formation varies greatly in different regions,—for example, it is much greater on the internal (medial) side of the recess for the paraflocculus than on the external (lateral) side. In the latter situation in the rat, resorption of bone is active. Other locations of relatively greater activity in relation to the internal auditory meatus and cochlea have been identified, sufficient to warrant the statement that the apparently excessive appositional bone formation in the vitamin-A-deficient animal also is correlatable with the pattern and rate of formation of the normal of the two species. The normal pattern of this region in the rat is somewhat different from that of the dog; likewise the untoward consequences of the deficiency in the two species.

Both in rat and dog, the replacement of cancellous bone by compact bone, which occurs in normal growth and remodelling sequences, ceases. This feature of the deficiency is particularly well exhibited in the bones of the dogs' calvarium and vertebrae.

In the chick (age of experimentation from day of hatching to three or four weeks), I have been unable to find any continuation of appositional bone growth other than a slight degree of generalized appositional bone growth of all bones. The general picture is that of greatly reduced bone growth, resulting in cessation of endochondral bone growth, great diminution of trabecular bone and of cortical bone, and great retardation of compact bone formation.

In no situation have I found evidence of untoward pressure effect upon the nervous system, resulting solely from local, apparently excessive, appositional bone growth.

Before further consideration of the thesis that all *appositional* bone growth in vitamin-A deficiency is the result of the carrying out of a normal growth pattern, an account

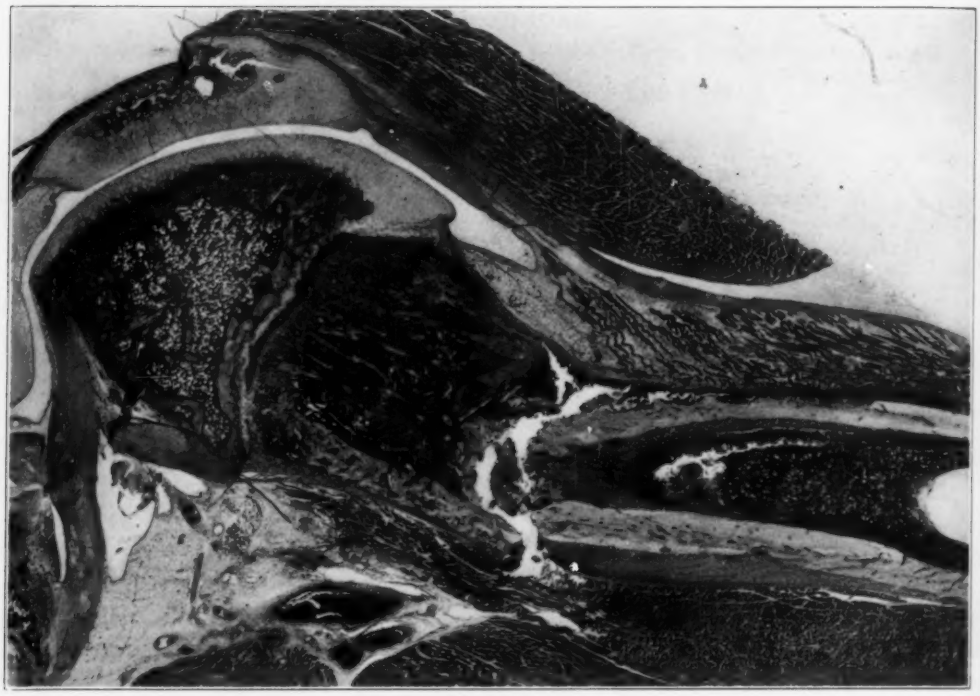

FIG. 12

Fractured femur near distal epiphysis of a rat which received 1250 international units per gram of weight of pure vitamin A for seven days following weaning at twenty-one days of age.

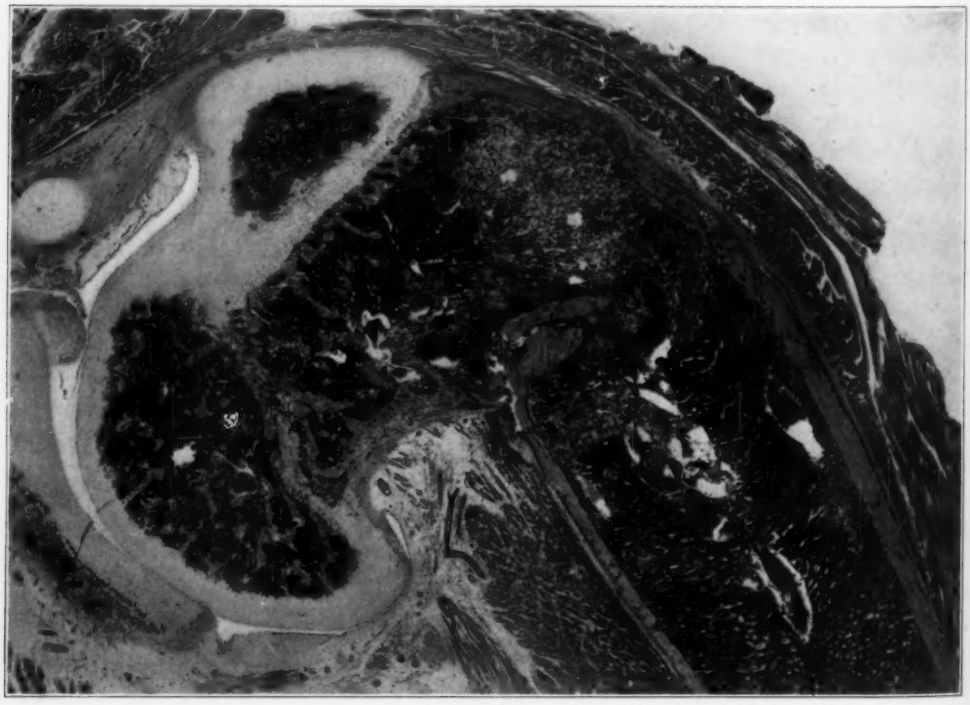

FIG. 13

Fracture of upper end of femur of a guinea pig which received 600 international units per gram of weight for twenty-four days. Initial and final weights, 100 and 109 grams. Ascorbic acid was given (60 milligrams, daily) for the last eleven days of the experiment. Note closure of the epiphyses and site of fracture in a region of active remodelling in normal growth.

of the consequences of excessive vitamin-A administration upon growth of young animals—rats, guinea pigs, and dogs—is necessary.

HYPERVITAMINOSIS A

Fractures of the long bones of rats following the administration of excessive amounts of fish-oil concentrates, presumably the result of the vitamin-A content, were reported by Collazo and associates in 1929 [19]. A brief review of the subject has been written by Wolbach and Bessey [19], who also reported that fractures of the bones of weanling rats could be produced in from six to eleven days by the excessive daily administration of pure vitamin A. No complete explanation of this disastrous effect upon growing bones was given, but we did write: "An hypothesis we hold at the present time is that the early consequence of excessive vitamin-A administration is the acceleration of some processes of bone growth, notably: (a) periosteal proliferation, (b) epiphyseal-cartilage sequences preliminary to endochondral bone formation, and (c) remodelling of bone attended by osteoclasis".

Continuation of the study of hypervitaminosis-A effects upon bone growth has been made possible through the assistance of Dr. O. A. Bessey of the Public Health Research Institute of New York, Dr. P. R. Howe of the Forsyth Dental Infirmary, Dr. D. M. Hegsted of the Harvard School of Public Health, Mr. Thomas Van Metre, medical student, and Dr. Charlotte Maddock, Research Fellow in Pathology.

The following résumé of observations and conclusions is based upon the study of many rats and guinea pigs. Many chicks, a few mice, and two puppies were also studied. Because the growth pattern of young chicks is quite different from that of the mammals studied, and because too few mice and dogs were used, this account is based upon the experiments with rats and guinea pigs. *However, in all species, the results of excessive*

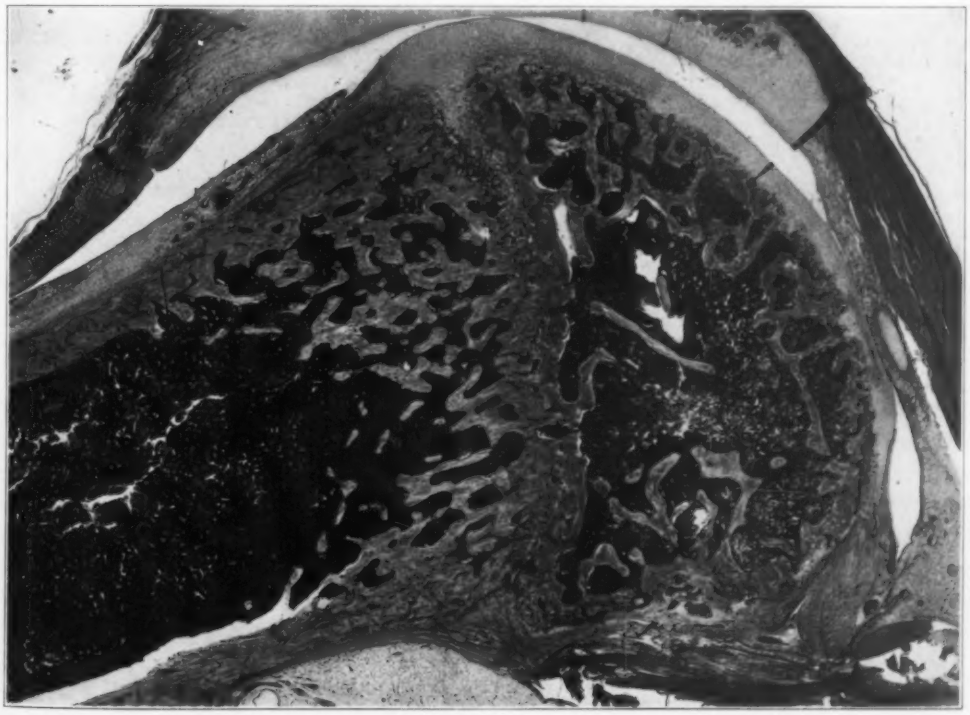

FIG. 14

From distal end of femur of guinea pig, illustrating early closure of the epiphysis. From nine days of age, this guinea pig received approximately 600 international units of vitamin A per gram, plus 120 milligrams of ascorbic acid for eighteen days, at which time it was killed. The large amounts of ascorbic acid did not influence the effect of the excessive vitamin A administered.

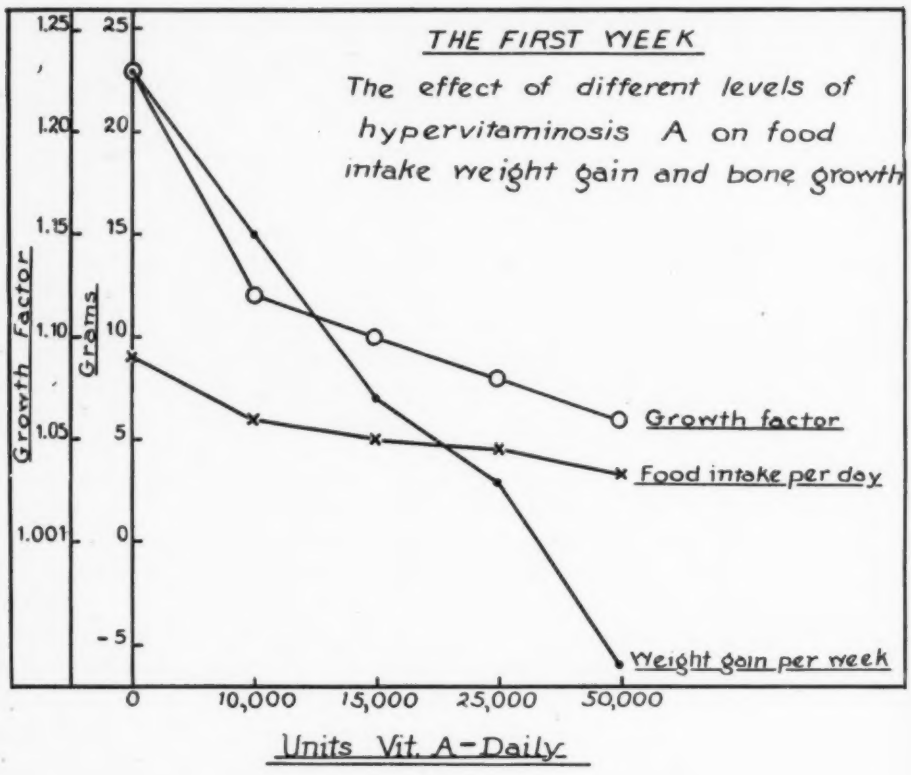

FIG. 15

Curves to show the relation of dosage of vitamin A to growth of a long bone (tibia), food consumption, and weight. All observations were upon weanling rats, twenty-one days of age; approximate weight at this age is forty grams. All measurements of bone were made from x-ray films, made daily. The values of growth given represent the percentage linear growth during one week. These curves are of little value except for documentation of the statement in the text, that rate of remodelling induced by vitamin A is independent of linear growth of bone.

vitamin-A administration are the same when correlated with the normal growth patterns of the bones, and can be precisely expressed in terms of accelerated sequences normal to the species and, in general, are the same as those of rats and guinea pigs.

In rats and guinea pigs there is great acceleration of all processes of bone growth which can be followed microscopically,—specifically, epiphyseal-cartilage sequences and the resorption of bone and appositional bone formation necessary in the remodelling of bone in skeletal growth.

In both species, fractures of long bones are produced in six to ten days, somewhat earlier and more frequently in rats than in guinea pigs (Figs. 12 and 13). The explanation of this difference may reside in the fact that in guinea pigs the epiphyses close, whereas in the rat many epiphyseal cartilages persist throughout life. In both species there is greatly accelerated maturation and degeneration of the epiphyseal-cartilage cells with concurrently rapid replacement by bone. In guinea pigs given excessive amounts of vitamin A (1000 to 1250 international units per gram of weight) from the age of ten days, the distal epiphysis of the femur and the proximal epiphysis of the tibia can be made to close in from ten to fifteen days—or at twenty to twenty-five days of age (Fig. 14). These epiphyses normally close after thirty and sixty weeks of age respectively [20]. In the rat, the adult marrow zone of quiescent epiphyseal cartilage may be attained in the same time period.

It was not difficult to prove, by means of serial sections of long bones (femur and tibia and fibula), that the accelerated remodelling processes were responsible for the production

of fractures; nor was it difficult to prove that the accelerated sequences, though disastrous in their consequences, were in complete conformity to normal growth patterns in character and localization. My first assumption was that the rate of remodelling was a function of linear growth. This logical rationalization had to be abandoned in light of the fact that in guinea pigs the remodelling sequences leading to fractures were in progress concurrently with the rapid consumption and ossification of the epiphyseal cartilages. Comparisons of the growth rates of the long bones of normal rats and litter mates receiving excessive amounts of vitamin A, by means of x-ray films were undertaken. The results showed clearly that in the first week, which covers the period in which fractures usually occur when large amounts of vitamin A are given, linear growth rate of bone is retarded, together with a decrease of food consumption and growth of the rat as a whole. The larger the amounts of vitamin A administered, *the greater is the retardation of growth and the greater is the rate and frequency of fracture* (Figs. 15, 16-A, 16-B, 16-C, and 16-D). The microscopic sequences tally perfectly with the gross changes. There is no evidence of increased rate of multiplication of cartilage cells; in fact, in both rats and guinea pigs, mitoses are fewer than in normal controls. Common to both guinea-pig and rat epiphyseal-cartilage sequences is the accelerated rate of growth, maturation, and death (cytomorphosis). The conclusion is warranted that an agent contributed by the dying or death of this specialized tissue—which maintains, until maturity of vertebrates, sequences essential to growth most commonly exhibited in the embryo and foetus—is responsible for the remodelling process of bone growth. Therefore, we may discard the simpler explanation that bone remodelling during growth is a response to shifting mechanics of the skeleton and musculature, and take recourse to the subtler processes revealed by experimental embryology and connoted by the term *induction*. In the present instance, epiphyseal cartilage is the *organizer* or *inductor*; the bone that undergoes resorption with concurrent appositional growth in conformity to a pattern is the *competent* tissue.

FIG. 16-A FIG. 16-B

FIG. 16-C FIG. 16-D

Four roentgenograms illustrating the effects of large *versus* relatively small doses of vitamin A upon the tibiae of rats.

Fig. 16-A: Normal 28-day-old rat.

Fig. 16-B: From 28-day-old rat which received 1250 international units per gram of weight for seven days prior to sacrifice.

Fig. 16-C: Tibia of a rat which received 250 international units per gram of weight from twenty-one days to sixty-nine days of age.

Fig. 16-D: Same rat with treatment continued to eighty-fifth day of age. The length of the tibia is within normal limits. The striking features are the thinness of the shaft and the contours near the epiphyses.

No attempt is made here to give a full account of the microscopic details of the effect

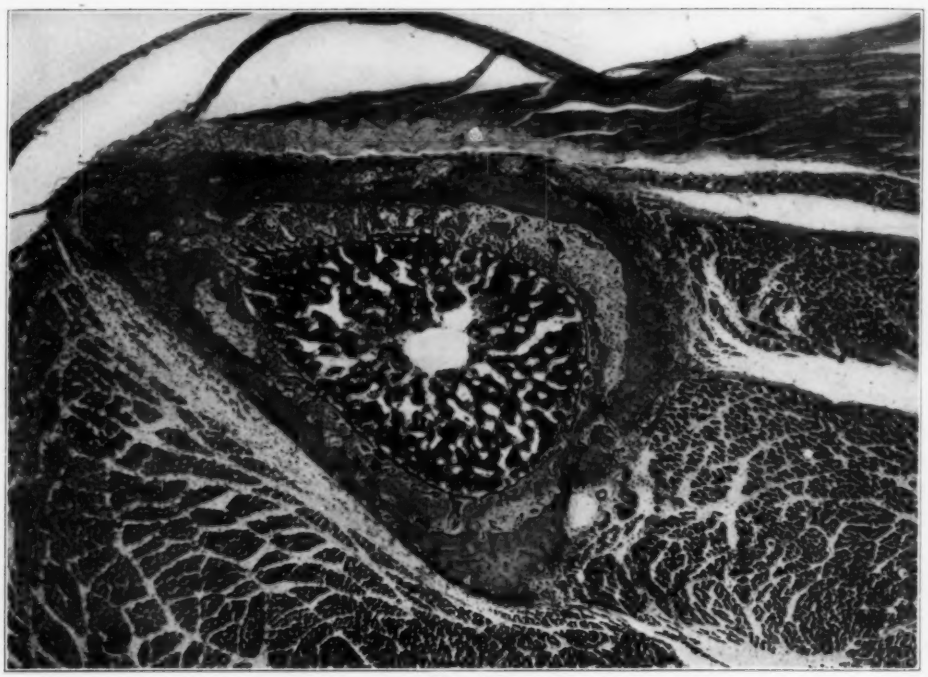

FIG. 17

Cross section of upper third of tibia of rat. This rat received 1250 international units daily from the twenty-first to the twenty-eighth day of age. Low-power photomicrograph, showing resorption of the cortex on the exterior, and newly formed partially calcified bone deposited internally. (See Figs. 18 and 19.)

already outlined. An intelligible brief description is possible, because no novel processes are involved and because the precise meaning of terms employed in descriptions of normal bone growth are applicable.

The Epiphyseal Cartilage

In guinea pigs and rats, the early change in the epiphyseal cartilage is the rapid maturation of the cartilage cells, so that the presence of vesicular cells extends far toward the epiphyseal side. Matrix production does not keep pace; and, in consequence, there is compression of the maturing cells on the diaphyseal side of the cartilage plate. There is deep and rapid penetration by capillaries, and often the capillaries in one column send off shoots laterally into cells of adjacent columns, giving rise to tuftlike patterns, similar to those seen in the initial periods of repair of experimental rickets. In guinea pigs, the acceleration of maturation results in complete consumption of the cartilage in ten to fifteen days and, as osteoid production keeps pace with the vascular penetration, ossification of the epiphyseal cartilage (Fig. 14). *The rate of change varies with the dosage of vitamin A.* Calcification of cartilage matrix apparently progresses with corresponding rapidity, and superfluous trabeculae on the diaphyseal side are consumed by osteoclasis. The indifferent or undifferentiated cartilage which lies on the epiphyseal side of the growth cartilage becomes in part calcified and disposed of by osteoclasis and in part removed by capillary penetration into dead cells and ossification.

In the rat, the same speeding up of cytomorphosis takes place, possibly sustained for a brief period by multiplication of cells in the proliferative layer. I have not been able to find any evidence of an increased rate of multiplication; in fact mitoses seem fewer in number than in the normal. In the course of a few days, cell division ceases and, the cells continuing to mature and degenerate and to be replaced by bone, causes the epiphyseal cartilage to become greatly reduced in width (Fig. 12). Always, however, in the period covered by our experiments, there persist groups of crescentic discoid cells of the prolifera-

tive layer and, on the diaphyseal side, globular cartilage cells arrested early in the process of maturation. Comparison of this late or final stage of the epiphyseal cartilage in hypervitaminosis A with the epiphyseal cartilage of arrested bone growth of the rat (a) in old age and (b) in inanition resulting from (1) inadequate amounts of a normal diet, (2) riboflavin deficiency, and (3) pyridoxine deficiency, discloses that the final result of the accelerated cartilage sequences in the rat conforms to the normal in amount and character of the epiphyseal plate that remains after cessation of growth. Concurrently with the acceleration of the epiphyseal cartilage sequences, there is a great acceleration of remodelling sequences, involving absorption of bone and appositional bone formation. The effects are easily visible after two days of administration of 1250 international units per gram weight, always restricted, however, to the regions where remodelling normally takes place. In general, in the long bones, the important remodelling process is concerned with the reduction of cross-sectional diameters and, therefore, the resorption of cortical bone is from without (periosteal surface) and the new formation is from within (endosteal). Resorption is by osteoclasis, the histological details of which are precisely those of osteoclasis in normal growth. The number of osteoclasts present is far in excess of that in normal remodelling of bone. The number of osteoclasts increases with the rate of resorption, determined by the dosage of vitamin A. New-bone formation in its histological details also corresponds precisely with normal appositional bone growth. Where remodelling is symmetrical—for example, at the upper end of the tibia—the result is essentially the production of two concentric layers,—an inner of newly formed bone, and an outer of the eroded remains of previously formed cortical bone (Fig. 17). In about the mid-portion of the femur, the pattern is an eccentric one, because the remodelling, which is necessary on the outer aspect of the shaft in relation to its linear curvature and to the prominent third trochanter, is not neces-

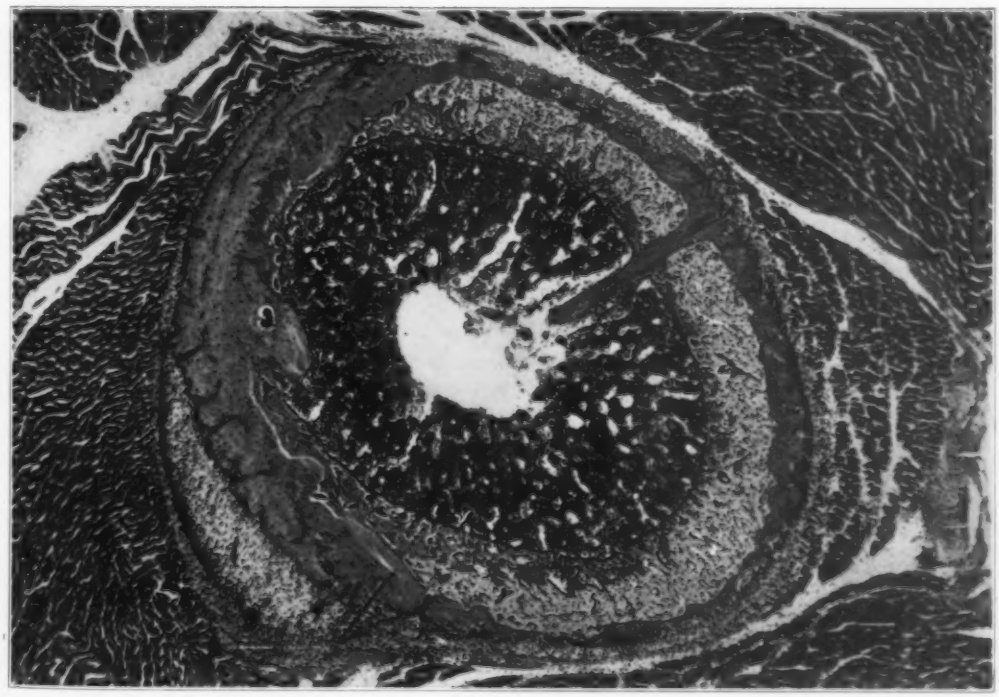

FIG. 18

Cross section of the humerus of the same rat in about mid-position. Notice the absence of much change in about one third of the circumference of the cortex. The cortex not undergoing remodelling is from a region which in normal growth remains unchanged. If a femur of a rat is rotated on its long axis, there will come to view, on the postero-internal surface, a straight outline of about the middle third of the shaft.

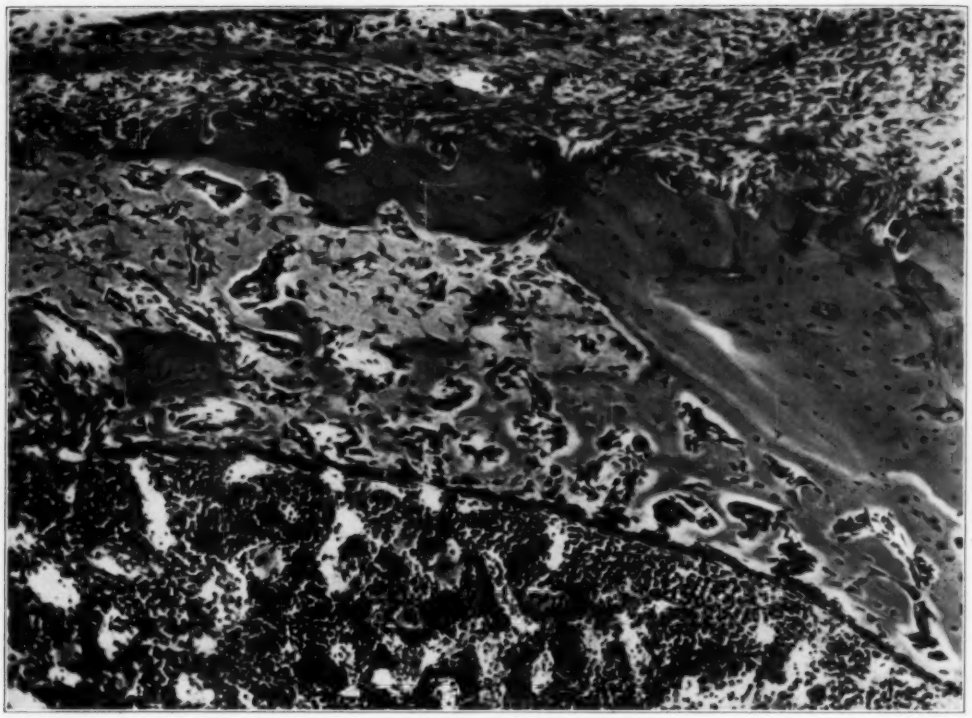

FIG. 19

A high-power detail of Fig. 18, showing resorption of exterior of cortex and newly deposited bone internally.

sary on the inner aspect of the shaft, which is not linearly curved for a length of six to eight millimeters of its mid-portion (Figs. 18 and 19).

Fractures of long bones are easily accounted for by the extensive loss of previously formed cortical bone before the newly deposited bone has acquired firmness sufficient to meet mechanical requirements. They occur at sites where remodelling in normal growth is most active. The microscopic appearances of the newly formed bone indicate that its lack of firmness is the result of two failures,—one of the matrix to mature, analogous to the collagen changes in cicatrix formation in general and exhibited in part by shrinkage, and the other to incomplete calcification, exaggeratively expressive of the lag in calcification seen in normal appositional bone formation and exhibited by the line of non-calcified matrix or osteoid, where normal growth is in progress.

The fibula of the rat, as it grows, not only becomes more widely separated from the tibia, but swings to a more posterior position. In hypervitaminosis A, the remodelling may be so rapid that at the upper end this bone may, in the guinea pig as well, lose all its pre-existing structure. The pattern of remodelling conforms precisely with the shifting relations of tibia and fibula which, by virtue of cement lines, can be equally well demonstrated in the normal fibula.

Another as yet incompletely studied result of hypervitaminosis A in the rat, dog, and guinea pig is the accelerated formation of compact bone which, as is the case with the chick, is most easily demonstrated in the bones of the skull, including those of the calvarium.

In the normally rapidly growing incisor teeth of rats and guinea pigs, the sequences of enamel and dentine formation are unaffected; perhaps they are somewhat accelerated. As growth of teeth is a one-way process,—that is, no remodelling occurs—the effects of the hypervitaminosis A are histologically inapparent.

In the chick, our studies are incomplete in regard to the effects of hypervitaminosis A.

Our experiments have included only the period from hatching to the thirty-second day. The problem in chicks has been complicated, because at hatching the replacement of cartilage in the shafts of long bones is far from completed. At present, an acceleration of endochondral bone formation and earlier establishment of the epiphyseal cartilage plates are the main results established. Acceleration of remodelling sequences can be seen, but has been inadequately studied because of lack of knowledge of the normal. The formation of compact bone in the vertebrae and bones of the skull is greatly accelerated.

The question of whether or not the action of vitamin A upon bones is a direct one upon the cartilage concerned with growth or operative through any of the ductless glands has led to the study of these organs—the pituitary gland, thyroid, parathyroids, and adrenals—both from A-deficient animals and from those exhibiting maximum effects from excessive vitamin-A administration.

In the excess-vitamin-A animals, I have found no changes demonstrable by ordinary techniques in pituitary gland, thyroid, and parathyroids. The adrenals do undergo changes, of moderate degree in the rat, outstanding in the guinea pigs. These changes include increase in size, loss of lipoid in the cortex, and a marked increase in mitosis of the cells of the zona fasciculata. In the guinea pig, the zona glomerulosa becomes atrophied. Also in the guinea pig, mitosis often remains incomplete, the chromosomes become fragmented and disperse. These effects are those known to result from inanition produced by underfeeding[2,14]. The difference in degree of the responses in rat and guinea pig noted in hypervitaminosis A also applies to the adrenals of the underfed of these two species.

In vitamin-A-deficient animals, the thyroid undergoes atrophy. The pituitary gland and parathyroids seem to be unaffected. The adrenals of guinea pigs long maintained in the deficiency show the same changes, though in much less degree, as those from animals with excess vitamin A.

In the rat, guinea pig, and chick the massive dosage of vitamin A we have employed is incompatible with life. A prompt effect is decreased food intake, followed by loss of weight, and lesions of the skin, notably of the eyelids, in the neighborhood of the nostrils and finally of other parts of the body. The skin lesions histologically are not unlike those of some vitamin deficiencies, notably of the heat-stabile vitamin-B group. Histological study reveals no lesions which can be held accountable for death. Certain changes in liver cells, rarely resulting in necrosis of individual cells, can be regarded as indicating that this organ is damaged directly, or equally well may be interpreted as the result of excessive vitamin-A storage. Metabolism studies on a broad front are plainly indicated for a search of heightened metabolisms. Calcium and phosphorus metabolism studies by methods usually employed have revealed no departure from the normal in rats, guinea pigs, and dogs. The phosphatase may be slightly increased, but, in view of the histology of the bone, is probably of no significance in elucidation of accelerated bone-growth sequences.

In young puppies receiving 300 international units per gram of weight, the cholesterol and phospholipid blood levels remain normal until late in the experiments, when severe consequences are manifest, at which time there is a significant drop both in cholesterol and phospholipids.

In guinea pigs the terminal values of cholesterol and phospholipids are increased to more than double the normal.

Thus far, in rats and guinea pigs we have obtained no appreciable amelioration of the "toxic" effects of vitamin A or arrestment of accelerated growth sequences by the administration of greatly excessive amounts of vitamin C and the vitamins of the B-complex group.

SUMMARY AND CONCLUSIONS

Vitamin A is essential for the activities of epiphyseal-cartilage cells, without which they cannot carry out the sequence of growth, maturation, and degeneration essential in

the mechanism of endochondral or replacement bone growth. Vitamin-A deficiency suppresses epiphyseal-cartilage-cell sequences and hence endochondral bone growth. Remodelling sequences, involving concurrent resorption of bone with bone deposition and replacement of cancellous bone by compact bone, cease to operate. Appositional growth of bone of periosteal origin continues, until inanition supervenes, *at a rate in conformity to the normal growth pattern in each site. Skeletal growth as a whole ceases.*

Excessive vitamin-A administration accelerates in general those growth sequences of bone retarded by the deficiency. It causes rapid consumption of epiphyseal cartilage and, in appropriate species, greatly premature closure of epiphyses; also, excessive rapidity of remodelling processes unrelated to linear growth of bones, but *definitely related to rate of epiphyseal-cartilage-cell cytomorphosis.* Both the rates of remodelling processes and of cartilage-cell sequences are quantitatively related to the amounts of vitamin A administered. Substitution of compact bone for cancellous bone in conformity to normal growth patterns is greatly accelerated. The fractures and sites of occurrence are fully explained by acceleration of remodelling. Bone which does not require remodelling for maintenance of the normal growth pattern is not responsive to the excess vitamin introduced.

Application of the facts presented will explain easily and completely the results of vitamin-A deficiency upon any of the simpler bony structures of the skeleton. In the case of the skull, the problem is a complicated one, because of the intimate relations of the individual bones to one another in the absence of joints, and because of the many epiphyses or sites of endochondral bone growth and directions of such growth in the bones comprising the base of the skull. However, here too the changes in bones at the base of the skull, in-

FIG. 20

FIG. 21

Fig. 20: Dissection of the optic tract of a normal litter mate, control puppy for comparison with Fig. 21, made from a vitamin-A-deficient puppy. Both of these dogs were thoroughly injected with formaldehyde immediately after sacrifice, so that the central nervous system was thoroughly hardened *in situ.*

Fig. 21: Vitamin-A-deficient litter mate of puppy of Fig. 20. Notice the tortuosity of the optic nerves in the orbit; also, compression deformities of cerebrum and cerebellum. The tortuous optic tract is evidence of failure of dimensional increase of the bony orbit in a region which excludes deformation by pressure of bone.

cluding the petrous bone, are precisely correlatable with normal growth patterns. The remodelling of the calvarium presents an interesting problem, because the increase of cranial capacity is the result of area increase of the enclosing bones. Growth at the base in bones of cartilaginous origin ceases. The bones of the calvarium of membranous origin normally increase their areas by appositional bone growth at the sutures. Change in radii of curvature also takes place as the cranial capacity increases. In the normal, these two features of growth—area increase of individual bones and changes in curvature—result in a pattern of growth of the membranous bones, in which there is resorption of bone on the exterior of the skull and deposition of bone on the interior surface. This remodelling of flat bones of the skull is suppressed in vitamin-A deficiency, as well as the remodelling of bones of cartilaginous origin, and results in thicker bones, cancellous in structure. One illustration of the fact that the growth of the skull as a whole is suppressed is the fact that in rats and dogs the extracranial lengths of the optic nerves are tortuous, because they become longer than the size of the orbits require (Figs. 20 and 21).

We may conclude that the pattern of bone growth is determined by epiphyseal-cartilage activities for which vitamin A is as essential, apparently without the intermediation of other organs, as it is in vision. We may deduce that in the maturation of the cartilage cells there is produced an inductor factor which is responsible for the maintenance of remodelling of bone during the growth period. Because the accelerated remodelling processes caused by excess of vitamin A adhere precisely to the normal growth pattern, bone which disappears during growth may be referred to as competent bone.

Recent crude experiments in which finely comminuted epiphyseal cartilage from calves has been injected into young rats have yielded results which indicate the correctness of the deduction that epiphyseal cartilage is the source of a factor or factors governing the remodelling of bone during growth.

The effects we have obtained do not duplicate in several ways those produced by excessive vitamin-A administration; nevertheless, they are quite distinctive and unlike those of any other agent affecting bone growth. Briefly, they are the continuation of normal epiphyseal-cartilage sequences, premature resorption of the trabeculae of the primary spongiosa, and, therefore, failure of incorporation of trabecular bone into cortical bone near the epiphyses, and great resorption of cortical bone at sites of remodelling. Osteoclasts are much less numerous than in excessive vitamin-A experiments, and appositional bone formation is not accelerated. Such bones have fractured in the course of handling the rats.

While the cartilage-injection experiments have yielded results of the general nature of acceleration of remodelling, important features of the normal processes are lacking, conceivably because labile factors have been lost in the preparation and preservation of the cartilage prior to injection.

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MOBILIZATION OF METACARPOPHALANGEAL JOINTS

Arthroplasty and Capsulotomy *

BY MAJOR SAMUEL BENJAMIN FOWLER
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Experiences in World War II have shown that stiff metacarpophalangeal joints are very common among patients admitted for reconstructive surgery of the hand. They follow most severe wounds of the hand and frequently injuries higher in the arm, particularly those wounds which are complicated by osteomyelitis, nerve injury, or large soft-tissue defects. Splinting has been responsible for the stiff joints in a number of cases.

ARTHROPLASTY

In the series of cases upon which this report is based, all the patients requiring arthroplasty presented destruction of one or both joint surfaces. This destruction was secondary to compound comminuted fractures directly involving the joint, except for one case where ankylosis was secondary to septic arthritis following a human bite. The inter-

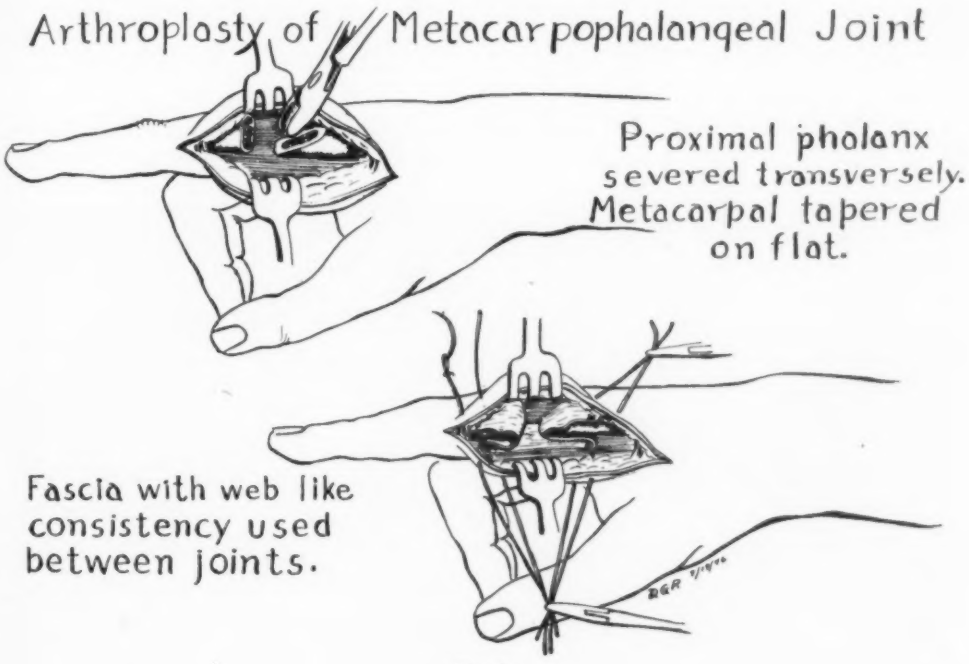

Arthroplasty of Metacarpophalangeal Joint
Proximal phalanx severed transversely. Metacarpal tapered on flat.
Fascia with web like consistency used between joints.

Fig. 1

phalangeal joints were generally stiff to some degree, and the intrinsic muscles were functionless in several cases because of destruction or adhesions. Some infection had probably been present in every case.

Arthroplasty is indicated for ankylosed metacarpophalangeal joints with less than 30 degrees of motion in a useful arc, if mobilization of the joint will restore a practical degree of function to the digit. Arthroplasty of the thumb has not been considered wise in any case of this series and has been done only once in the little finger. If a digit is permanently

* Presented at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 22, 1946.

FIG. 2-A

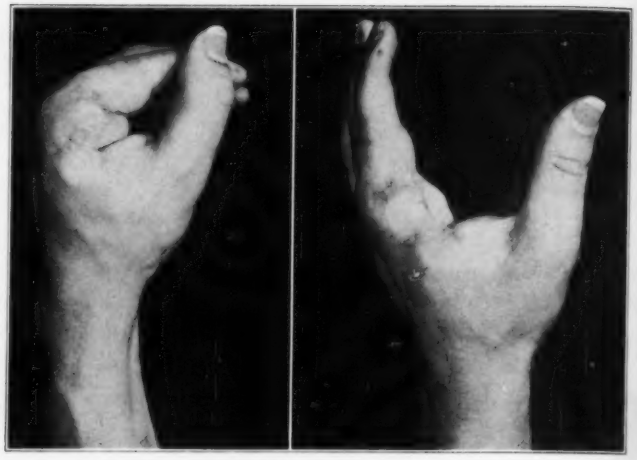


FIG. 2-B

Fig. 2-A: Case N. H. Photograph showing amount of flexion in hand before surgery. The index finger had previously been amputated.
Fig. 2-B: Photographs showing motion in the metacarpophalangeal joint of the middle finger after arthroplasty.

anaesthetic, is deformed, or lacks flexor tendons, an arthroplasty will not improve the function of the hand much, no matter how good a joint is made. If there is excessive shortening of the metacarpal, arthrodesis may be indicated.

Technique of Arthroplasty

An incision, approximately five centimeters long, is made over the dorsolateral aspect of the joint. The joint is exposed through the space between the interosseus and extensor ten-

FIG. 3-A

Case J. W. Roentgenograms showing ankylosis of metacarpophalangeal joint of the middle finger. Note also fracture of proximal phalanx of the index finger, causing limited motion in that digit.

dons. A joint space of about one centimeter is obtained, largely at the expense of the proximal phalanx. The metacarpal head is fashioned so that it inclines somewhat volarward, is tapered sharply anteroposteriorly, and is left broad and flat laterally. The phalanx is cut transversely. The fine fascia overlying the fascia lata on the anterolateral aspect of the distal third of the thigh is used as the interposition membrane. It is meticulously interposed between the joint surfaces, beneath the extensor tendon and deep to the tendons of the interossei. If there has been a loss of intrinsic muscle substance, it is important to transfer the flexor digitorum sublimis tendon into the lateral bands of the extensor aponeurosis. The shortening of the ray which results from the injury and which is exaggerated by arthroplasty may so weaken the intrinsic muscles that they cannot forcibly flex the metacarpophalangeal joint. Theoretically, it might be wise to transfer the flexor digitorum

FIG. 3-B

FIG. 3-C

Fig. 3-B: Photographs of hand, showing range of motion in the fingers before operation.
Fig. 3-C: Photographs showing function in hand following arthroplasty.

FIG. 3-D

Postoperative roentgenograms, showing arthroplasty of metacarpophalangeal joint of the middle finger.

sublimis in every case; but actually it is necessary only if the intrinsic muscles are damaged or the metacarpal is unduly shortened.

Immediately after the operation, a cast is applied with traction to the affected digit, with the metacarpophalangeal joint flexed about 60 degrees. In about seven days, the patient begins to exercise the joint. Three weeks after operation the cast is removed, and the exercises are increased. If there is resistance to full flexion, a removable traction splint, which flexes the affected joint, is applied. It is worn part time with diminishing constancy, but is continued as long as flexion is incomplete, even if it be an additional two months.

Results

Sixteen arthroplasties were done on thirteen patients. In nine patients, at least 70 degrees of active motion was obtained; in two of these, the flexor digitorum sublimis was transferred to the lateral bands, because of complete destruction of the interossei. In the four remaining patients, conditions were briefly as follows:

Case 1. There was ankylosis of all the joints of the fingers, with lacerated volar digital nerves and flexor tendons. Arthroplasties and nerve sutures were done on three fingers. Patient now has 30 to 40 degrees of motion in the metacarpophalangeal joints, and will have tendon grafts.

Case 2. Patient has 90 degrees of passive motion, but, due to a severed extensor tendon, he lacks 40 degrees of complete extension, actively.

Case 3. Arthroplasty of the index finger, and capsulotomies of the middle, ring, and little fingers were performed. Patient obtained 60 degrees of active flexion in the index finger and approximately normal function of the others.

Case 4. All joints of the index and middle fingers were stiff in extension, following compound fractures which involved the metacarpophalangeal joints, with anaesthesia of the fingers, and no flexor-tendon activity. After arthroplasties, the man had about 40 degrees of motion in the index finger and 30 degrees in the middle finger. He can now touch the tips of his fingers to his thumb.

The metacarpophalangeal joint of the fingers is probably the most favorable joint in the body for arthroplasty. The joint is primarily concerned with mobility, is not subject to the trauma of weight-bearing, and is under complete muscle control in all directions. After arthroplasty, the joints are stable and relatively frictionless. Ankylosis does not recur following surgery, if good muscles are acting directly on the joint, and if flexion is maintained for an adequate period. There have been no postoperative infections in this series, and the function of the hand has been improved in each case.

CAPSULOTOMY

Stiff metacarpophalangeal joints with intact joint surfaces present a more varied and subtle problem. They are always fixed in extension or hyperextension, because the shape of the metacarpal head and the attachment of the collateral ligaments are such that the ligaments are tight in flexion and relaxed in extension. If these collateral ligaments are allowed to remain in a relaxed state for a few weeks, bathed in oedema fluid or inflammatory exudate, the fibers are literally glued together, the ligaments are shortened, and flexion of the joint is effectively blocked. However, the pathological process does not stop here. The volar portion of the head of the metacarpal is not now functioning as a joint surface; it is in intimate contact with the tight anterior capsule and becomes glued to the capsule after a few weeks. Fibrous connective-tissue union follows, and the cartilage may be replaced over the volar surface of the metacarpal head; this further blocks flexion. At this stage, if the collateral ligaments are removed, normal flexion is not restored, but the joint becomes the hinge type, so that it flexes by opening like a book, instead of by the phalanx gliding around the metacarpal head.

Shortening of the collateral ligaments, followed by fixation of the anterior capsule to the metacarpal head, constitutes the essential mechanical change that occurs in a metacarpophalangeal joint which is held in extension in the presence of local swelling. The

FIG. 4-A

Case W. C. Roentgenograms of hand, upon admission.

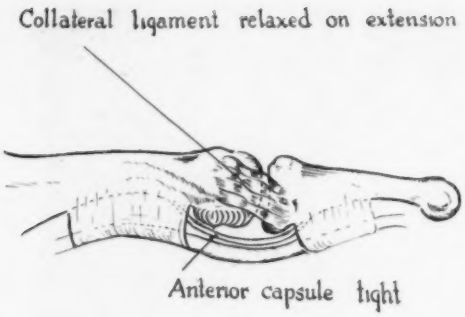
FIG. 4-B

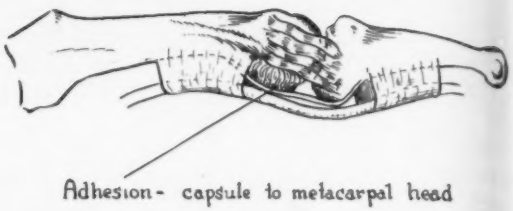
FIG. 4-C

Fig. 4-B: Photographs showing motion in hand before surgery. Active motion in the index and middle fingers is limited because of the stiff ring finger.

Fig. 4-C: Photographs showing motion in hand following arthroplasty of the metacarpophalangeal joint of the ring finger and amputation of the little finger.

degree to which these changes become irreversible depends on the local tissues and their nutrition. The factors which cause the metacarpophalangeal joints to be held in extension must be carefully evaluated. Splinting of these joints in extension by the surgeon does occur, but it is not generally the predominant factor, for these patients are young, and such immobilization is not often persevered in too long. However, in the presence of certain pathological processes, it may act as the precipitating cause. The most important factor is muscle imbalance, due to paralysis of the intrinsic muscles, or any condition of extensor muscles or tendons which does not allow complete flexion of all the joints of the finger. The lumbricales and interossei are the only muscles which directly flex the metacarpophalangeal joints. When they are functionless, the extensors are not sufficiently opposed in their action on this joint; and extension deformity is apt to result, particularly





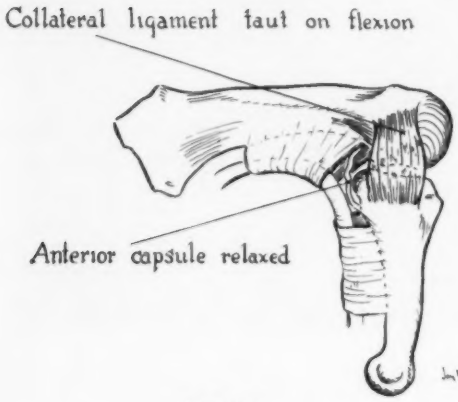


FIG. 5-A

Showing collateral ligaments of the anterior capsule in a normal joint.

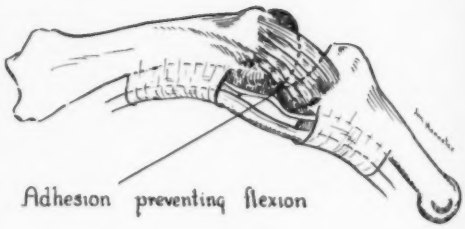


FIG. 5-B

Showing the mechanism of limited flexion in a stiff metacarpophalangeal joint where joint surfaces are intact. Flexion is prevented because of short collateral ligaments and adhesion of the anterior capsule.

in the presence of swelling and in the absence of proper splinting. The same situation exists in a metacarpal fracture with bowing which tightens the extensors and relaxes the intrinsic muscles by shortening the distance between their origin and insertion. Being short small muscles with a low amplitude of excursion, the interossei and lumbricales do not adapt themselves well to shortening of the distance between their origin and insertion, as do the long forearm muscles. Flexion contracture of the wrist tightens the extensor tendons and causes them to overbalance the intrinsic muscles. Extensor tendons which are tight, due to adhesions or pathological changes in the muscles, are common and are a difficult problem. Severe wounds of the forearm are generally splinted, with the wrist and fingers extended, so as to approximate destroyed muscles or tendons. It is necessary and proper to extend the wrist but, if the fingers are held extended in a patient where tendon graft or transfer is later indicated, they will be held in such a position by adhesions. At least one extra operation is necessitated to obtain flexion, and even then the end result of tendon transfer or graft is likely to be compromised. Poor nutrition in the hand, with resulting vessel and nerve lesions, greatly accelerates the pathological changes in the joints and makes them much more resistant to treatment.

Capsulotomy is indicated on metacarpophalangeal joints fixed in extension, if the joint surfaces are intact and if they cannot be mobilized with physical therapy and traction. In the patients of this series, traction was not continued beyond three weeks, if there was no inclination of the joint to yield. Capsulotomy is frequently done in connection with other operations,—such as nerve suture, bone graft to metacarpals, tendon transfers, skin grafts, and arthrodesis of the wrist. It cannot be successfully combined with any operation which necessitates extension of the metacarpophalangeal joint following surgery, or with a flexor-tendon graft. Extreme caution must be exercised if a capsulotomy is done

FIG. 6-A

FIG. 6-B

Fig. 6-A: Case J. S. Showing loss of flexion in metacarpophalangeal joints of index and middle fingers.

Fig. 6-B: Showing motion in fingers following capsulotomy of metacarpophalangeal joints and lysis of extensor tendon to the index finger.

on a hand with circulatory insufficiency, for gangrene of the fingers may result. The operation will not be successful, unless the extensor tendons are free and allow complete flexion. The ankylosis is apt to recur, if muscle imbalance or metacarpal deformity remains.

Technique of Capsulotomy

An incision, about 1.5 centimeters long, is made in the dorsal web on both sides of the affected joint. A one-centimeter longitudinal incision is made in the extensor hood, 0.5 centimeter lateral to the extensor tendon. The anterior part of the extensor hood is then retracted, with the intrinsic tendons and the collateral ligament completely removed. This is done on both sides of the joint, according to the technique of Bunnell[1]. The joint is then flexed by placing the thumb over the dorsum of the base of the proximal phalanx and pushing the phalanx forward and flexing it at the same time. If the interphalangeal joints extend as the metacarpophalangeal joint flexes, it indicates that the extensor tendon is caught in scar tissue and this must be corrected. If the joint opens like a book instead of the phalanx gliding around the metacarpal head, the base of the phalanx is being blocked by an adherent anterior capsule. The joint can hinge open, so that the phalanx is at right angles to the metacarpal; but, unless the base of the phalanx passes around to the anterior portion of the metacarpal head, the deformity will certainly recur. The adherent anterior

FIG. 7-A

FIG. 7-B

Fig. 7-A: Case J. P. Photographs showing loss of flexion in metacarpophalangeal joint and loss of extension in interphalangeal joints of index finger, following old, compound fracture of second metacarpal with destruction of intrinsic muscles to index finger.

Fig. 7-B: Showing function in index finger following capsulotomy of the metacarpophalangeal joint and transfer of flexor digitorum sublimis tendon to extensor aponeurosis.

FIG. 8-A

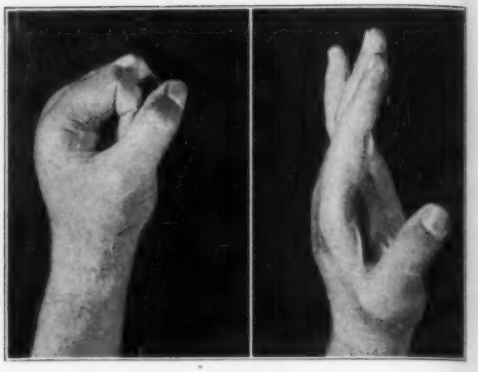


FIG. 8-B

Fig. 8-A: Case C. B. Received severe injury to hand, with traumatic amputation of the middle finger. A pedicle flap had been applied, but had healed poorly. Fingers were stiff, with the metacarpophalangeal joint in hyperextension.

Fig. 8-B: Following capsulotomy of all metacarpophalangeal joints. Function is considered excellent in view of the extreme scarring of the hand.

capsule is often stripped from the metacarpal head, when the joint is forcibly flexed; if not, it may be reflected from the metacarpal head with a blunt dissecting probe. Occasionally, following capsulotomy, the extensor tendon will become subluxated to the side of the joint when it is flexed. If this condition is allowed to persist, the metacarpophalangeal joint cannot be actively extended. It may be corrected by sectioning part of the lateral expansion of the extensor tendon on the side of the subluxation or by tightening the opposite side. If the latter manoeuvre is used, flexion should not be started for one week and should be obtained gradually by traction. Following surgery, the metacarpophalangeal joints are held in flexion, either by rigid splinting or by traction. If it is necessary to release the

FIG. 9-A

Case R. M. Roentgenograms of hand before surgery.

FIG. 9-B

FIG. 9-C

Fig. 9-B: Photographs showing function in hand with ununited fracture of second metacarpal and stiff metacarpophalangeal joint.

Fig. 9-C: Photographs showing function in index finger following bone graft to second metacarpal and capsulotomy of the metacarpophalangeal joint.

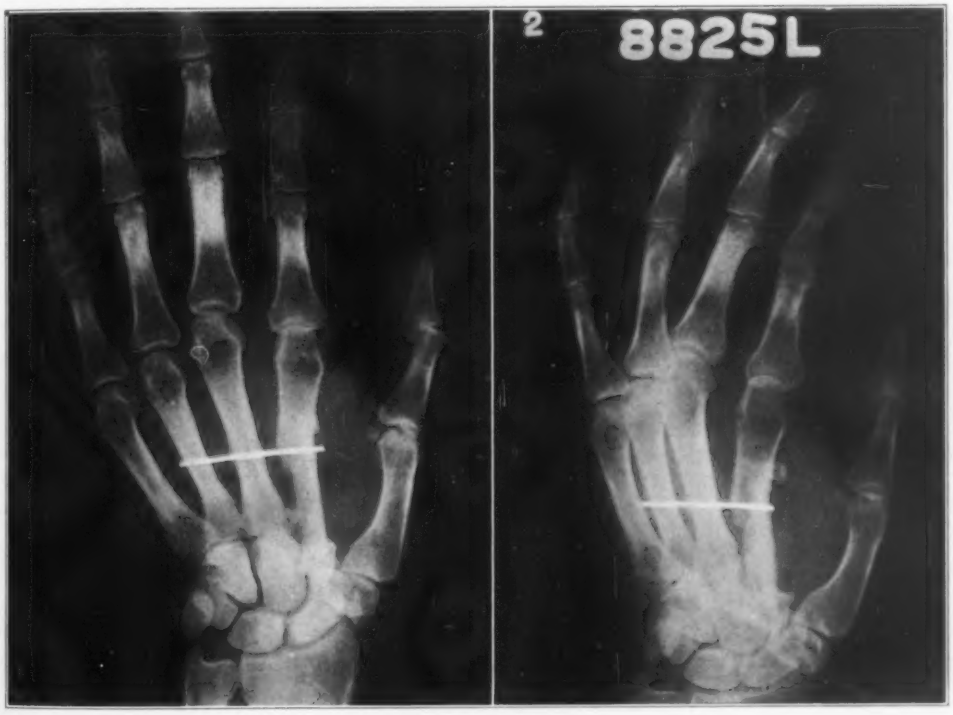


FIG. 9-D

Roentgenograms of hand following bone graft to the second metacarpal.

splint, because of pain or undue swelling, a traction cast is reapplied at the earliest opportunity, and correction is gradually obtained. Full correction is maintained for three weeks; then a removable traction splint is applied, which is worn for about one month and removed gradually. It is reapplied if there is a tendency to recurrence of the deformity.

Results

The results from capsulotomy are excellent, and 80 to 90 degrees of motion may be confidently expected, if local tissues are good and the mechanics of the hand are satisfactory. Over 100 capsulotomies have been done, mostly in conjunction with other operations. The associated pathological conditions have been such that meticulous tabulation

of the end results involves too many factors for a brief presentation. Factors contributing to imperfect results are:

1. Failure to excise adequately the collateral ligament, particularly the extreme anterior portion;
2. Failure to strip the anterior capsule from the metacarpal head;
3. Failure to keep the joint flexed long enough;
4. Subluxation of the extensor tendon;
5. Muscle imbalance in the hand;
6. Ununited and malunited metacarpal fractures;
7. Poor condition of the local tissues, including cicatrix and deficient circulation.

CONCLUSIONS

Arthroplasty and capsulotomy are relatively simple procedures which can be used to mobilize metacarpophalangeal joints of the fingers, affording useful joints, unless there are too many other pathological changes.

The poor results of capsulotomy and arthroplasty are due to conditions outside of the joint itself, which often can be corrected by associated surgical procedures.

1. Bunnell, Sterling: Surgery of the Hand. Philadelphia, J. B. Lippincott Co., 1944.

DISCUSSION

Dr. Sterling Bunnell, San Francisco, California: This excellent paper is entirely in conformity with my points of view, and with what has been carried out in the nine Army General Hospitals where the work of reconstructing hands has been done. I have reviewed Major Fowler's fine work in repairing hands in the Baker General Hospital; I am familiar with his results, and can substantiate his statements.

For both capsulotomy, which is really capsulectomy, and arthroplasty of the proximal finger joints, there are certain requirements. There must be redundancy of dorsal skin and the surrounding parts must be in good condition. The muscles must be working. The long extensor tendon must be free from adhesions, and there must be a strong flexor. Normally the intrinsic muscles flex these joints; but, in combined median and ulnar palsy of these muscles, many patients with their long flexors can flex these joints to a right angle but weakly, though not enough to prevent claw-hand. Therefore, if the interosseus muscles have been destroyed, we must substitute for them in one of two ways. Either the tendon of the flexor sublimis digitorum is transferred to the dorsal lateral band in the finger, or the long flexors are given a better angle of approach to the proximal segment of the finger. To obtain this better approach, the pulley over the metacarpal head is slit laterally.

The hood over the head of the metacarpal may be of thin deep fascia or of paratenon from over the fascia lata. It should prevent the four surrounding tendons from adhering to the head of the metacarpal. Correct muscle balance is also necessary. If upset by angulation forward of the forearm bones, wrist, or metacarpals, the tightened extensor tendons will not allow the proximal finger joints to flex.

It is essential to maintain this flexion, at first by plaster and then by elastic splinting. Three types of splints for this purpose have been described elsewhere,—one of plaster and one of metal, each with an outrigger over which run rubber bands; and, also, the versatile knuckle bender splint.

THE DEFINITION OF HUMAN LOCOMOTION ON THE BASIS OF MEASUREMENT

With Description of Oscillographic Method * †

BY R. PLATO SCHWARTZ, M.D., AND ARTHUR L. HEATH, B.S., ROCHESTER, NEW YORK

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The importance of normal locomotion to the physical and economic advantage of every individual is self-evident. The significance of human locomotion to orthopaedic surgery is found in that large percentage of patients who require treatment to prevent disabilities in the function of walking.

In previous papers[8,9,10], we have presented a review of the literature on this subject. In the relatively few references, some of which must be regarded as classics[1,2,5,6,7], there was none which could be applied to clinical problems. Work done in the past did not provide the means for a quantitative definition of normal human locomotion; therefore, functional disabilities and the results of treatment could be recorded only in words and by various methods of illustration.

FIG. 1-A

Six pressure-sensitive discs are applied to respective levels on the plantar surface of each foot.

FIG. 1-B

Subject, oscillograph, and operator. Records are made with the subject either wearing shoes or barefoot.

The nature of the problem has defined the narrow limits within which a combination of particular resources can be made to provide records, from which human locomotion can be reduced to measurement. This has been repeatedly emphasized in the successive efforts which we have made since 1926. It is these exacting requirements which have delayed the development of the present resources for a period of eight years.

* Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

† Read before the Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 27, 1946.

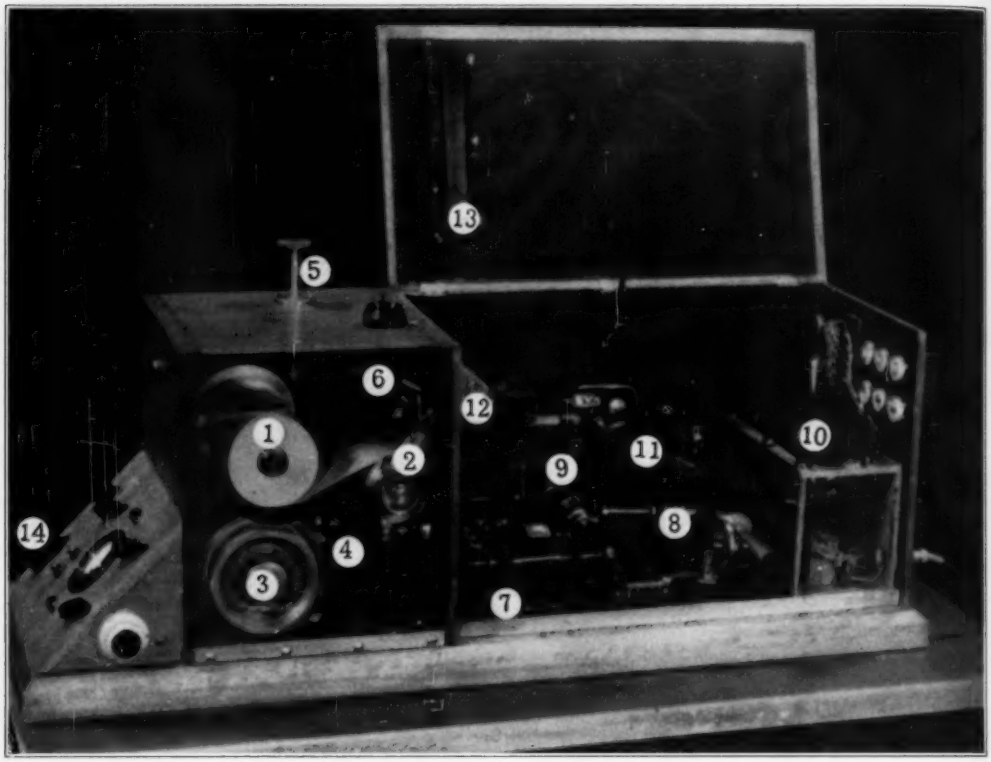

FIG. 1-C

Mechanism of oscillograph.

1: 200-foot roll of recording photographic paper.
2: Driving roller.
3: Take-up chamber.
4: Cut-off knife.
5: Cut-off knife control.
6: Record-numbering projector.
7: Synchronous motor.
8: Timing disc.
9: Magnetic clutch.
10: Galvanometers.
11: Viewing mirror.
12: Prisms and collimating slits.
13: External viewing screen.
14: Control panel.

In 1932 we were mindful of what the most useful records should reveal. On a background of time in seconds and tenths of seconds, there should be recorded the sequence, duration, and amount of pressure, as expressed through the plantar surface of each foot in the act of walking. At that time, however, most earnest efforts failed to provide two essential pieces of equipment,—(1) galvanometers with required characteristics, and (2) material for the construction of pressure discs which had to be precise and uniform in their reaction.

In 1939 an instrument was designed and constructed in the laboratory, which has been in continuous use for recording normal and abnormal locomotion since that time. Provision was made in 1941 for simultaneously recording locomotion and muscle function on the same record.

The instrument is an oscillograph, with independent light sources for each of two sets of six prisms which reflect light to corresponding galvanometers. These galvanometers are electrically connected to six respective areas on the plantar surface of the subject's feet (Fig. 1-A) by a twenty-five-foot flexible cable (Fig. 1-B). Within, the mechanism is operated by a synchronous motor which drives the timing disc and the rollers, which move the two hundred feet of photographic paper at a constant rate of speed. Light beams reflected from the galvanometer mirrors expose the paper through a slit in the camera box. The exposure of the moving photographic paper to the light beam from the swinging

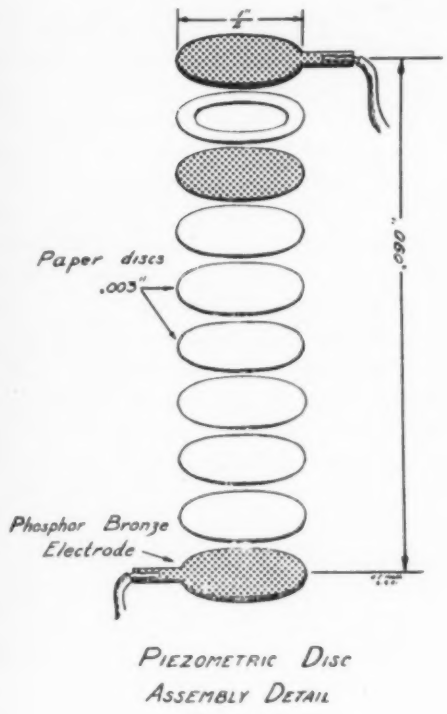

FIG. 2-A

Arrangement of carbon discs in construction of piezometric (pressure-sensitive) disc.

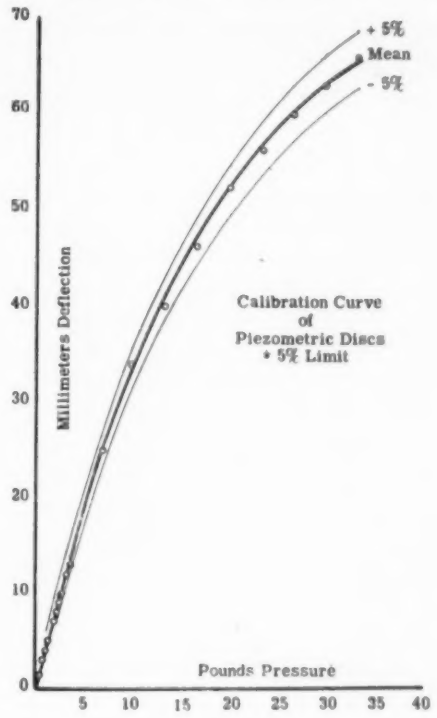

FIG. 2-B

Each piezometric disc is made to conform to this calibration curve.

mirror causes a curve to be photographed upon it. Each successive record is photographically numbered and a light-tight take-up chamber is provided for removal of exposed paper (Fig. 1-C).

The pressure-sensitive discs are constructed as indicated in Figure 2-A. All twelve discs are made and calibrated to react uniformly to successive amounts of pressure as in the distribution curve (Fig. 2-B). They are applied to the respective areas on the plantar surface of each foot, as in Figure 1-A, and effectively secured with Scotch tape. These areas were not arbitrarily selected; by extensive tests they were found to produce the most valid records of the stance phase of normal locomotion. Moreover, it has been found that no significant

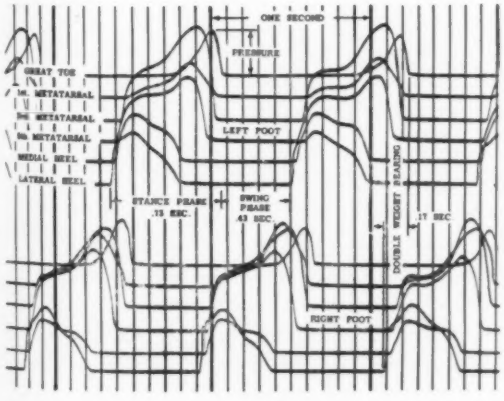

FIG. 3-A

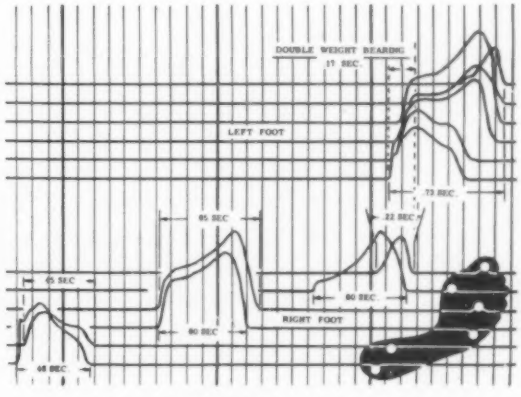

FIG. 3-B

Fig. 3-A: This record of "normal" barefoot locomotion on a background of time in seconds and tenths of seconds illustrates the source of measurements.

Fig. 3-B: This illustration is a break-down from Fig. 3-A to provide a better understanding of Fig. 3-A.

departure from normal function of the foot can prevail without recorded evidence of the difference. There has been no evidence of unfavorable psychological reactions, when these resources have been used to make clinical records of locomotion.

With the oscillograph in operation, "electrical foot-prints" are photographed upon the record, while the patient walks without instruction as to rate. A record of normal barefoot locomotion is illustrated in Figure 3-A. In Figure 3-B, a single stance phase of the right foot has been divided into its three major functional divisions. These respective divisions—reception, support, and propulsion—are revealed by the pressures and duration of weight-bearing on the lateral and medial aspects of the heel, the fifth and third metatarsal heads, the first metatarsal head, and the great toe.

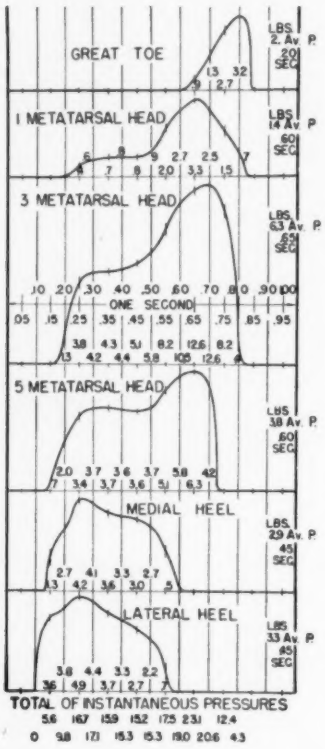

FIG. 4-A

In relation to the end of the third division of the stance phase (propulsion) on the right foot, there is the beginning (reception on the heel) in the stance phase of the left foot. Here we find recorded simultaneous weight-bearing on both feet. This is commonly referred to as the period of double weight-bearing. The functional pattern of the stance phase of the left foot duplicates the corresponding normal functional record of the right foot (Fig. 3-A).

We have found that the day-to-day variations in

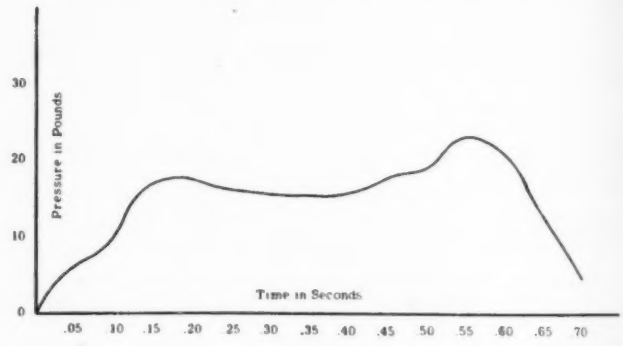

FIG. 4-B

Fig. 4-A: To illustrate the method of measurement, the respective curves were vertically separated from each other without alteration of time interval. Pressures are measured at each 0.05 second intervals for respective curves.

Fig. 4-B: A single line curve for the step shown in Fig. 4-A.

the oscillographic record of the same individual are not great, and this is resolved in the average for the individual. Also, variations between individuals lose their significance in the computation of a group average.

Biological data in general, regardless of the problem or method of measurement, are recognized as subject to wide ranges of individual variations. Differences found in relation to oscillographic records of locomotion fall within a useful range of constancy, as defined by the practical application of biological data in other fields of investigation.

The method of measurement for the quantitative expression of such functional records is illustrated in Figure 4-A. It is accomplished through the use of direct-reading scales. Pressures are measured at each 0.05 second interval for respective curves. Maximum and average pressure values for each of the curves are easily obtained from the tabulation; duration of pressure is read directly from the record.

For the gross comparisons of the function of the foot as a whole, advantage is gained by the use of the single line curve. This is constructed by addition of all pressure values

which occur simultaneously within the 0.05 second intervals from the beginning to the end of the stance phase of the step. These successive values are shown at the bottom of Figure 4-A,—that is, the total of instantaneous pressures.

When plotted on a graph, with the same time base as in Figure 4-A, the resulting single curve represents the summation of pressures common to the foot during the stance phase of the step. By thus resolving all functional details of the six most significant levels of the foot into a single curve, the most graphic quantitative comparison of variations in foot function is achieved (Figs. 10-A, 10-B, 10-C, and 10-D).

In Figure 3-B the duration of contact of the respective levels of the right foot with the floor is indicated. This is summarized in Figure 4-A. The period of double weight-bearing (Fig. 3-A) is 0.17 second, in which time interval the entire plantar surface of the left foot, not including the great toe, was in contact with the floor. This period of double weight-bearing is constant for normal individuals when walking at the average rate of two steps per second. The duration of double weight-bearing lengthens as the rate drops, shortens as the rate increases, and is absent in running (Fig. 9-C).

The swing phase of the step,—that is, from the time the right or left great toe leaves the floor until the right or left heel contacts the floor, is ± 0.5 second, as illustrated in Figure 3-A (0.43 sec.). The stance phase of the step is ± 0.7 second, 0.2 second longer than the swing phase.

All records of normal locomotion have essentially equal duration of time for the stance phase, double weight-bearing, and swing phase of the step for right and left foot, respectively. Any significant inequality of time interval for one or more of these recorded values is characteristic of a limp, and is accompanied by departure from the pressure curves which are typical of normal function.

It is common knowledge that an individual may be recognized by the sound of his footsteps. This means that each individual walks differently. However, it follows that all normal individuals superimpose their slight differences upon a functional expression which

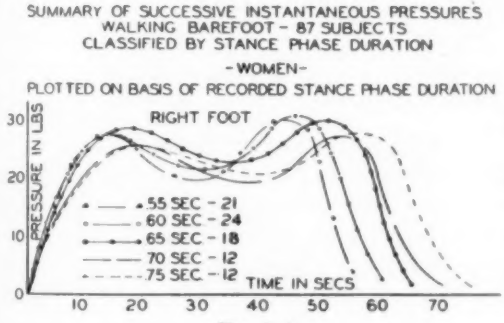

FIG. 5-A

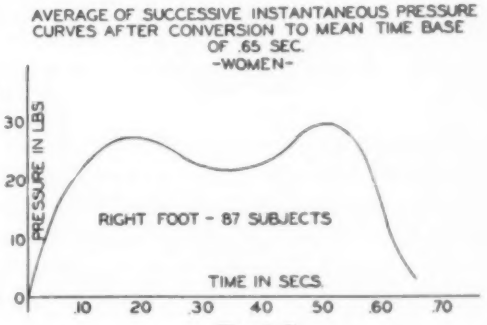

FIG. 5-C

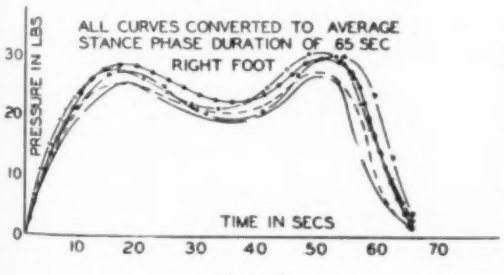

FIG. 5-B

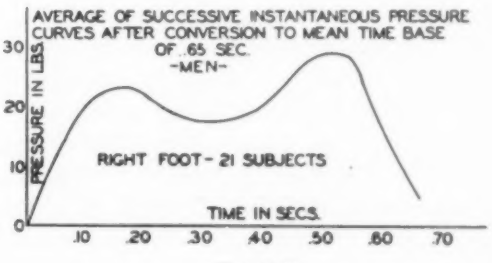

FIG. 5-D

Fig. 5-A: Curves representing the single line analysis of eighty-seven nurses.
Fig. 5-B: After the curves in Fig. 5-A were statistically converted to the standard time base of 0.65 second.
Fig. 5-C: The average pressure flow for the foot as a whole, for the entire group.
Fig. 5-D: Average curve for a group of male subjects resulting from the same method.

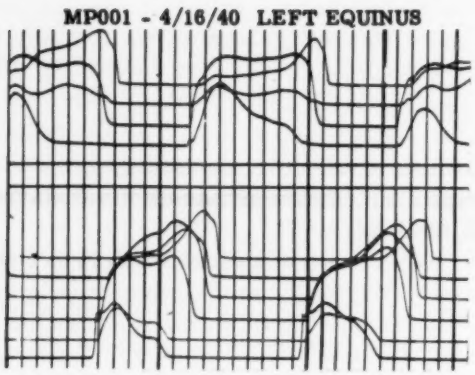

FIG. 6-A

No pressure on left heel.

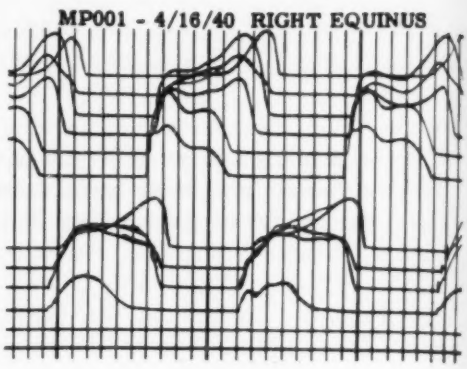

FIG. 6-B

No pressure on right heel.

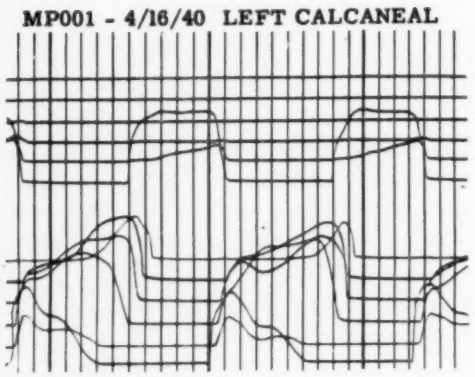

FIG. 6-C

No pressure on left medial heel and fore part of foot.

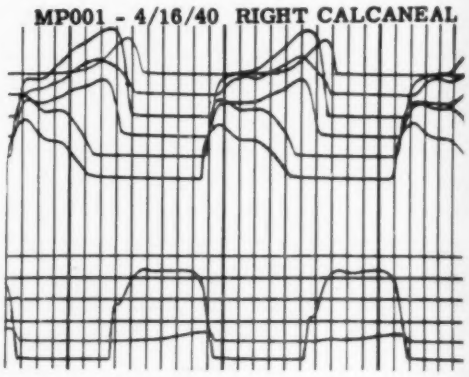

FIG. 6-D

No pressure on right medial heel and fore part of foot.

NOTE: Functional compensations required on "normal" side represent gross departures from normal function in each instance.

is common to all others in walking from place to place. For these reasons, we have found differences in the records of normal individuals. But the characteristics common to all dominate, and the individual characteristics are eliminated when the records of a large number of normal subjects are reduced to a single curve.

This fact was revealed by the following procedure. The records of eighty-seven nurses were divided into various groups on the basis of age, weight, height, rate of walking, and pronation. This was done for the purpose of determining which of these factors gave significant evidence of a dominant influence, and to determine the base line against which all records, normal and abnormal, could be compared. We have found that the rate of walking has a significant effect upon the pattern of the curves in the stance phase of the step. As yet we have been unable to fully explore for similar groupings on the basis of age, weight, height, and pronation.

The influence of rate of walking is revealed in Figure 5-A. The eighty-seven records were divided into five groups on the basis of the average duration of the stance phase in each record,—that is, the rate of walking. The method of measurement previously described was used, and the results in each of these groups are represented by a single curve. These respective curves express the total amount of pressure exerted through all of the respective pressure discs at successive time intervals during the stance phase of the step. The variations due to rate of walking are quite evident.

In Figure 5-B the measurements for respective groups were statistically reduced to a

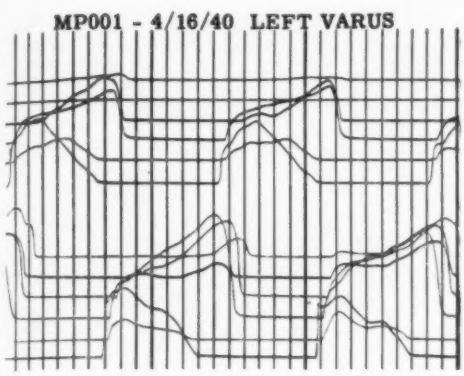

FIG. 7-A

Function absent on left medial heel, the first metatarsal head, and great toe.

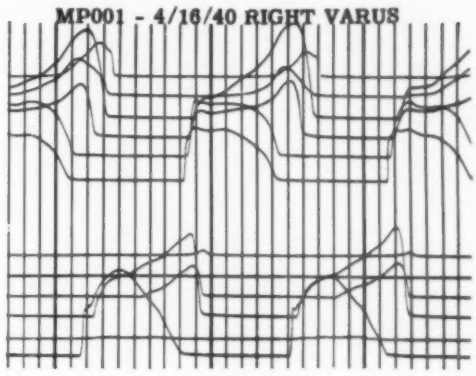

FIG. 7-B

Function absent on right medial heel, the first metatarsal head, and great toe.

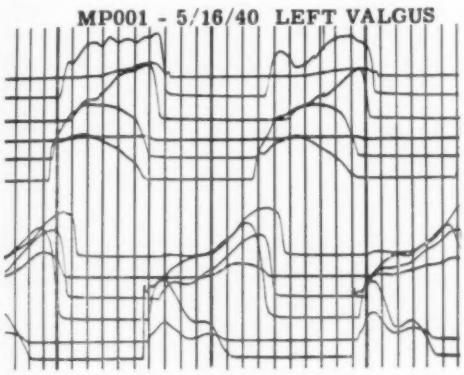

FIG. 7-C

Function absent on left fifth metatarsal head and great toe.

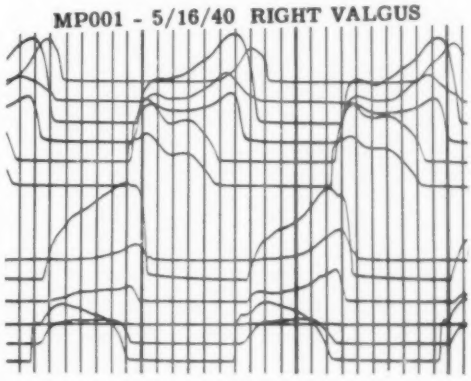

FIG. 7-D

Function absent on right fifth metatarsal head and great toe.

NOTE: Absence of function on one or more levels is accompanied by greater function on other levels of the same foot.

common duration for the average stance phase of the step,—that is, 0.65 second. It is evident that the removal of the variable in this instance was accompanied by a greater similarity in the curves representing respective groups. Here we have expressed a fair example of the high-limit and low-limit characteristics which are common to the single curve for normal locomotion. At present it is felt that a single curve, expressed in terms of 0.65 second for the duration of the stance phase, is the most reliable expression of normal locomotion.

The single curve (Fig. 5-C) represents the average of the five curves in Figure 5-B. Figure 5-D represents a similar analysis of records of twenty-one men.

The evidence thus far presented does emphasize the fact that human locomotion is a very complex function. Although it is normally expressed with ease, its simplicity is denied by any serious effort to quantitatively define its characteristics. Concurrent with such an effort, there is fostered a respect for the many factors which simultaneously prevail in varying degrees and, therefore, constitute variables which must be given due consideration.

In arriving at the quantitative definition of the normal characteristics of barefoot locomotion, we feel that the method of recording can be accepted as reliable. We are equally confident that the simple method of measurement of the records is free from significant errors in reducing (1) the rate to a common stance phase of 0.65 second, and (2) the respective six curves for each foot to a single curve.

When this identical procedure is applied to the quantitative evaluation of disabilities

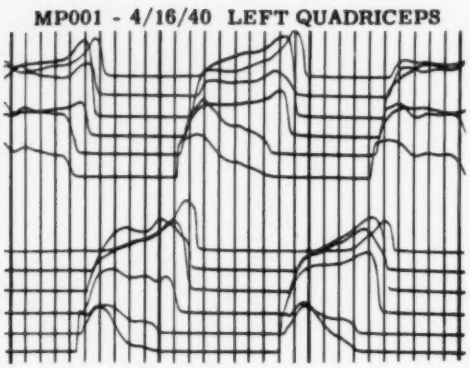

FIG. 8-A

Duration of stance phase longer on the left. Gross departure from normal curves bilaterally.

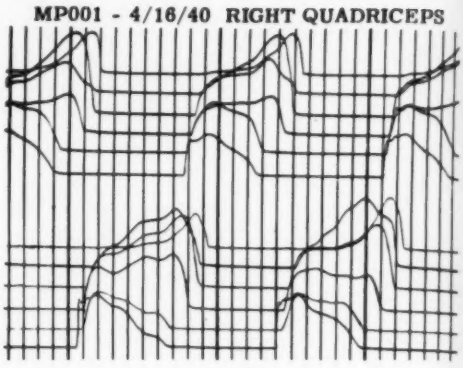

FIG. 8-B

Curves of right foot are essentially a mirror image of those for left foot (Fig. 8-A).

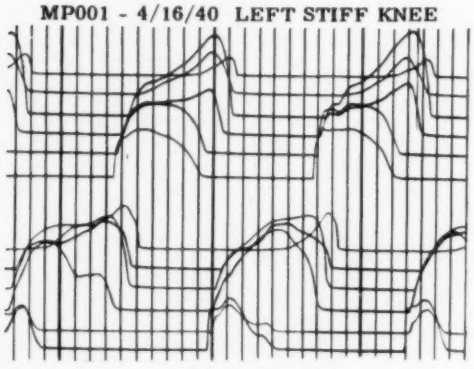

FIG. 8-C

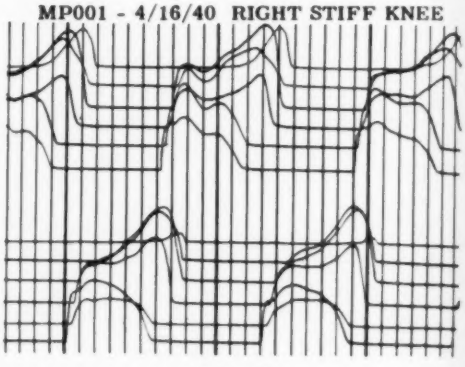

FIG. 8-D

Same characteristics in curves of left and right foot on respective records. Gross differences in compensatory function by left and right feet in respective records.

in locomotion, there is an expression of values very different from those which fall within the limits of normal function. This fact fosters further confidence in the quantitative definition of normal human locomotion, as previously presented.

It is not our purpose, within the scope of this paper, to discuss the details of recorded disabilities. However, the potential significance of information pertaining to normal locomotion may be indicated by a brief reference to the manner in which the records of disabilities differ from those of normal locomotion.

In Figure 6-A, a left equinus gait produced a record free from weight-bearing on either medial or lateral aspects of the left heel. The increased work done by the fore part of the left foot is indicated by the longer duration of the stance phase (slower rate) and the increased height of respective curves, particularly at the third metatarsal level. Essentially the same characteristics prevail in the record of a right equinus gait (Fig. 6-B).

A left calcaneal gait is illustrated in Figure 6-C. The weight is borne primarily on the lateral aspect of the left heel. In Figure 6-D, the record of a right calcaneal limp is essentially identical to that in Figure 6-C, the continuously straight base lines corresponding to the first metatarsal head and great toe.

Figure 7-A reveals the absence of pressure on these levels in a left varus limp. The lateral heel and fifth metatarsal curves are higher and longer; the medial heel and third metatarsal curves are lower than normal. In the record shown in Figure 7-B, there are revealed the characteristics of a more exaggerated varus limp of the right foot. Here there is omission of weight-bearing on medial heel, first metatarsal head, and great toe. The major

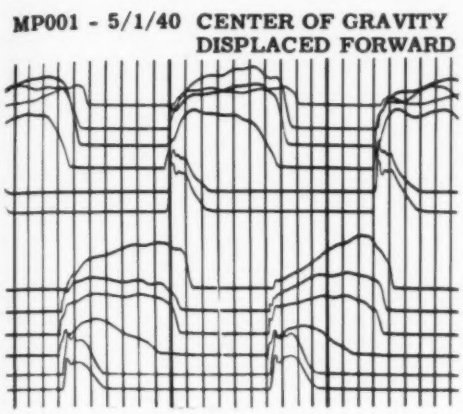

FIG. 9-A

Minimum function on heels, maximum function on fore part of the foot; duration of stance phase lengthened.

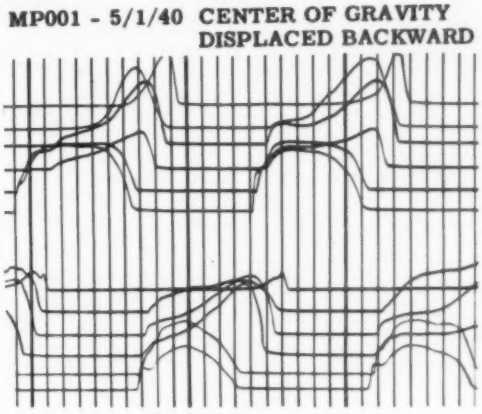

FIG. 9-B

Maximum function on heels, minimum function on fore part of foot; duration of stance phase lengthened.

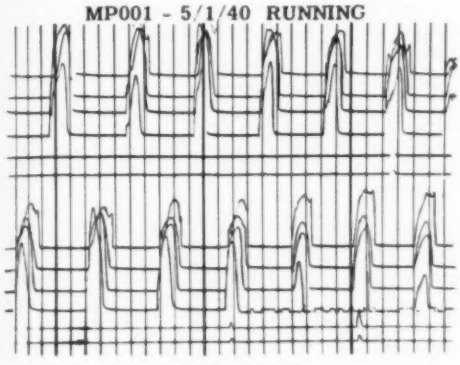

FIG. 9-C

Stance phase shortened to ± 0.15 second, without heels participating; ± 0.1 second between contact of respective feet.

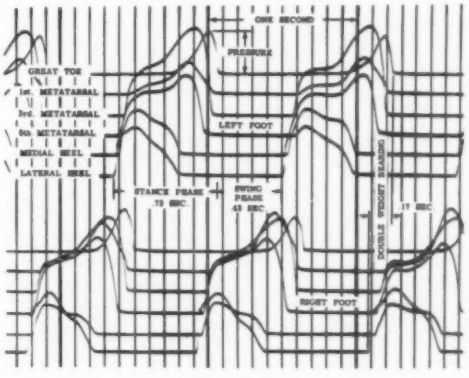

FIG. 9-D

Record of normal walking for comparison.

weight is borne on lateral-heel and fifth-metatarsal levels, with only a small share carried by the third metatarsal.

The recorded characteristics of a valgus limp are revealed in Figures 7-C and 7-D, left and right foot, respectively. The most exaggerated deformity is expressed in the functional deficiencies recorded in Figure 7-C. Partial or total absence of weight-bearing at the levels of the fifth and third metatarsals and great toe, with grossly elevated first-metatarsal curve, is characteristic.

The quadriceps limps recorded in Figures 8-A and 8-B, left and right foot, respectively, show significant function on all six levels of respective feet, low and abnormal distribution of pressures on heel and first-metatarsal levels, high and abnormal distribution of pressures on the other three levels.

The records of left and right stiff-knee limps (Figs. 8-C and 8-D) show a lessening of great-toe function on the affected side, and emphasize the functional adaptation required of the normal extremity to compensate for the functional disability expressed in a fully extended stiff knee.

With marked forward displacement of the center of gravity while walking (Fig. 9-A), a minimum of normal foot function is expressed through the heels, the fore part of the foot and great toes doing the work. These functional expressions through respective levels of the foot are essentially reversed, when the center of gravity is correspondingly displaced

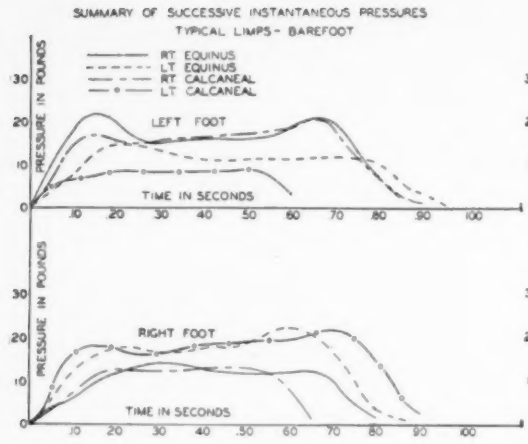

FIG. 10-A

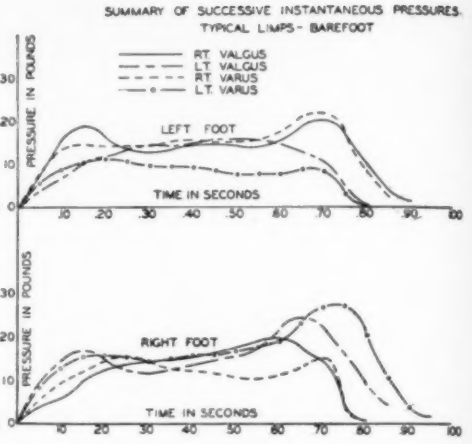

FIG. 10-B

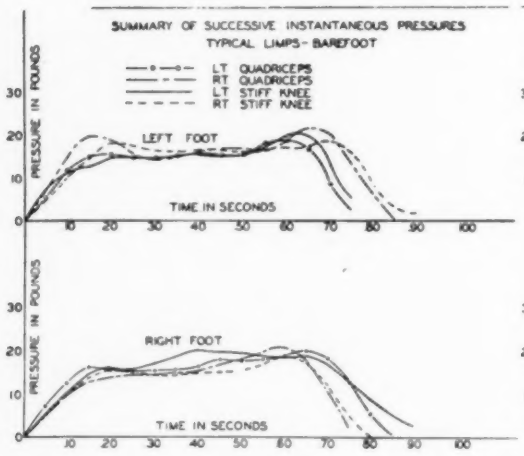

FIG. 10-C

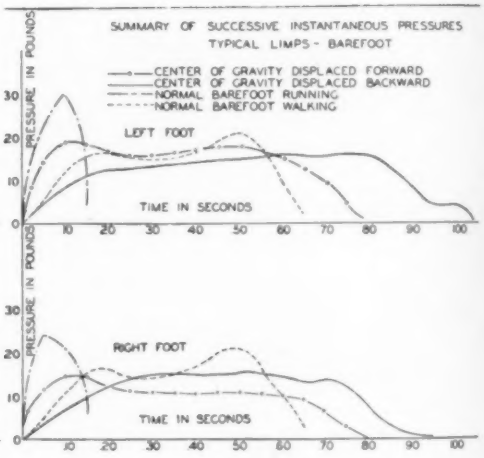

FIG. 10-D

backward (Fig. 9-B). In running (Fig. 9-C), the duration of the stance phase is reduced to 0.15 second ($\pm$ 0.7 sec. for normal walking) without any weight on the heel. Foot function is limited to the respective levels of the fore part of the foot, including the great toe. The record of normal barefoot walking (Fig. 3-A) is here repeated (Fig. 9-D), for ease of comparison with the records of abnormal limps.

In Figures 10-A, 10-B, 10-C, and 10-D, the respective records of functional disabilities have been reduced to single curves for respective feet. Such disabilities are characterized by the manner in which they alter the duration of the stance phase on the respective affected and normal extremities. For this reason, the records of functional disabilities should not be reduced to the 0.65 second for the duration of the stance phase of the step.

In all of these single curves of functional disabilities, it will be noted that the duration of the stance phase is significantly greater than normal, and that the curves are grossly different. This may be accepted as an index of our experience since 1940. From this we conclude that the variations within the limits of normal locomotion cannot produce a record characteristic of any significant functional disability.

SUMMARY

This paper may be regarded as a progress report on a study of human locomotion which has been under way for twenty years.

The reason for this study is that improvement in the treatment of dysfunction in other fields of medicine has followed in the wake of a quantitative definition of normal function.

By the application of special resources, herein described, human locomotion has been recorded. The normal characteristics have been measured, and the stance phase has been expressed in a single curve, as related to time and pressure.

Likewise, it has been shown that any significant dysfunction of human locomotion is characterized by a gross departure from the normal curve of the stance phase.

The application of these resources to clinical problems provides for the evaluation of therapeutic results on the basis of measurement instead of memory.

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DISCUSSION

Dr. Verne Inman, San Francisco, California: The present studies of Dr. Schwartz have resulted in an improvement over the previous methods by indicating the pressure transmitted through the various portions of the foot, as well as the time interval of contact. The piezometric discs are ingenious and, if they are as readily calibrated as indicated in the paper, the discs will certainly have a wider application. The lack of an adequate mechanism for the measurement of pressure has been a stumbling block to engineers, as well as to investigators in the biological field. I regret that Dr. Schwartz did not give more minute details of construction in his publication, but sincerely hope that he will make them available to other workers.

The present study shows the progressive transference of weight from the heel through the fore part of the foot in normal and pathological gaits. The *time* of transference of pressure through the selected parts of the foot is nicely shown. However, I should like to ask Dr. Schwartz if it is not true that only the pressures transmitted through the areas covered by the discs are recorded and not the total pressures of the foot, since other areas than those beneath the discs may be in contact with the floor. Furthermore, the pressure discs record only vertical forces, and it would be exceedingly interesting to know if sheers or torques are present as weight is borne upon the foot. To obtain these latter data, it seems to me, the use of a force plate would be required.

As Dr. Schwartz himself indicates, the study of human locomotion is complex, and I believe the full value of his work will not be appreciated until it is correlated with the motion of the other segments of the lower extremity. It is hoped that such investigations will soon be undertaken.

Dr. Robert W. Johnson, Baltimore, Maryland: In these few minutes, Dr. Schwartz has given the distillate of many long and tedious hours of experimentation and observation. I recall well a visit to his laboratory a number of years ago, where flashing lights revealed the earliest patterns of the tread mechanism of locomotion. Following through his series of articles, one sees the various difficulties ingeniously met; and it would seem that now he has definitely established the time sequence and pressure

variations on the sole of the foot, due to gait. Having established the normal, he is now beginning, as he has shown, to study abnormal gaits; and this offers an absolutely inexhaustible field for investigations with its analyses of the functional, as well as of the anatomical, disabilities arising from muscle imbalance and joint restrictions.

I certainly want to congratulate him on his tenacity, ingenuity, and ability for critical analysis. I also want to sympathize with him in the prospect of the tremendous volume of problems which this method puts in his lap for solution.

Dr. Arthur Steindler, Iowa City, Iowa: I always have had the greatest admiration for Dr. Schwartz's achievements in this very difficult line of observation of the human gait. In order that we may understand how it fits into long studies of human gait, we will have to realize that this represents a most ingenious, simplified method of measuring counterpressure by direct observation. That was the thing that Braune and Fischer failed to develop. For this reason, they had to follow a circuitous route of observing pressure by calculating the derivatives from path curves, to velocity curves, and to the curve of acceleration, which obviously will give the force, because acceleration is a function of the force. It took Braune and Fischer eight hours to dress their subject, in order to demonstrate with Geisler tubes the path curves alone. In order to bring about a determination of those horizontal components of the pressure curve, we are now in possession of enough information to calculate the horizontal components, as the heel is being put on the ground, as well as the strength of propulsion and restraint in the terms of this resultant component during the standing phase. The development of Dr. Schwartz's method has marked a tremendous advance in our ability to measure the force directly. The problem now is opened to the general benefit of the profession. It finally has become something that the orthopaedic surgeon can tackle.

Dr. Schwartz (closing): It has been our continuous desire to develop a method of recording locomotion that would have some potentially clinical usefulness. At present, it seems to us that we have accomplished this objective and reduced the method to a relatively simple procedure.

It is true that we do not measure total pressures. It is also true that total pressure has not been our aim. We feel that we have briefly given you the results that provide an index of the normal pattern of human gait.

Granted, there are individual characteristics of locomotion. One of the reasons for potential discouragement in the beginning was the oft-repeated statement that "there was no use in doing this work because everyone walks differently".

Such objection was not valid. Everyone has a common pattern of locomotion on which individual characteristics are superimposed. We have recorded and given quantitative expression of the common pattern.

I am duly grateful for the time and consideration which were given to this paper by Dr. Inman, Dr. Johnson, and Dr. Steindler, as indicated in their respective discussions.

PRE-EMPLOYMENT EXAMINATIONS OF THE BACK *

BY STEELE F. STEWART, M.D., HONOLULU, TERRITORY OF HAWAII

This presentation is the account of an attempt to prevent and to eliminate backache among men engaged in a heavy industry,—stevedoring. The types of backache will be reviewed briefly, and perhaps reclassified.

1. Referred backaches, resulting from abnormalities of the prostate, gall bladder, or other organ, do not come within the scope of this study.

2. Acute traumatic backache as the result of falls or direct blows may come to any man and, since they cannot be anticipated, do not fall within the field of preventive medicine or of this paper.

3. Acute atraumatic backaches are of two general types:

A. The first type may arise from several diverse but related causes,—such as fatigue, exposure, or salt deficiency.

Fatigue is produced (1) when a novice does unaccustomed tasks for a prolonged period (as Holman Day observed, "No one knows how many backaches are in an old stun wall."); (2) by uneconomical posture; (3) by sleeping as if suspended by one's head and heels in a sagging bed; or (4) by doing monotonous repetitive acts. It is this last cause which enters into most backaches in stevedores, which arise from handling cargo. For example, in working with sugar, each man picks up, carries, and stacks a hundred-pound sack of sugar every seventy-nine seconds, eight hours a day. With pineapples, each man handles a fifty-five-pound case of pineapples every forty-five seconds throughout the day. Stevedoring is therefore a very active occupation, and there is little opportunity for postural or static difficulties to develop. Hence postural peculiarities are not considered in this group.

Exposure is the second major cause of this type of backache. In the tropics, it may be the result of working with "icehouse" cargo or sitting in sweat-soaked clothing under the chilling effect of the balmy trade wind. This causal relationship was noted by Pasquin a century and a half ago, when he wrote: "He's lumbagoed [by the north or east wind] the rest of his days".

Salt deficiency is not at all uncommon in the tropics.

These causes may be present singly or jointly. In backaches from these causes, the muscles feel as though they had jelled; motions are slow and painful; the affected metameres are cooler than the adjacent ones; and the person tends to become limber with diverse activity. To relieve these backaches, the Polynesians have their children walk on their backs; personal experience has shown this treatment to be dramatically effective.

The problems presented by these backaches are in part medical, but for the most part they arise from the working habits of the men and the conditions imposed by the types of cargo and the places in which they work.

B. The second clinical type of acute atraumatic backache is usually attributed to a "sprain", arising from lifting anything heavier than a cigarette paper. The motions producing the backache are usually flexion and twisting. The condition is acutely painful and very disabling; hence it is called acute. In reality, it is usually chronic, because it is frequently episodic. It is to this group that the present study has been especially directed.

The author first encountered this problem in 1942, when he went to Honolulu and became associated with the Medical Group, an established firm which had been caring for about 850 stevedores for the Castle and Cooke Terminals, Limited. The first problem presented was backache in stevedores.

* Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 27, 1946.

This was an especially acute problem in the Territory at that time, for two reasons: First, in stevedoring, the laws, both federal and territorial, make no provision for compromise settlements in case of unapportionable disability arising from a pre-existing condition, such as hemivertebra or hypertrophic changes associated with sprain of the back. One such case has been carried by the company for more than four years. In other words, the company is responsible for care of the man. Second, the recently invalidated martial law likewise prevented the man from leaving his job, so that only death, the doctor, or crime could part him from his employer. It was, therefore, unwise to hire a man who might prove to be a liability, and equally unwise for the employee to try work for which he might prove unfit.

Backache was also a problem at the Navy Yard, among the civilian employees and other contract workers. Thousands of Caucasian and Negro workers were employed by defense contractors and at the Navy Yard; all of them were supposed to have had pre-employment examinations, but these must have been cursory.

The stevedore applicants were largely local men in their early thirties; two-thirds were Filipinos, a seventh were Japanese, and a fifth were of mixed races. These local residents had their own homes or boarding houses, and friends; and they ate their accustomed foods as far as possible. The individuals of a similar average age who had come from the mainland were strangers in a strange land, ill-housed, uncongenially quartered, poorly fed, separated from home and loved ones by censorship, irregular and slow communications, and lack of travel facilities; frequently they had no friend to whom to talk. Both groups were subject to the sunset-to-sunrise curfew and a strict blackout; they were restricted as to movement; entertainment was non-existent; the choice of food was limited; and all were "frozen" to their jobs. All these inconveniences bred unhappiness and ills.

Largely from these two groups, during the past four years the author has examined 560 persons with backache; 470 were men and 90 were women. During the first year, it was noted that roentgenograms of the back showed abnormalities in all but about 5 per cent. of the indviduals with backache. A program of pre-employment examinations was instituted for the stevedores of Castle and Cooke Terminals on April 23, 1943, and consisted of obtaining anteroposterior and lateral roentgenograms of the lumbosacral junction of all applicants. In the following three years, 1049 applicants for stevedoring were examined. A similar examination was made of 131 men, employed as stevedores before April 23, 1943, who complained of chronic backache. The group of private patients with backache, the complaining stevedores who had not had an examination of the back, and those who had had pre-employment examinations form three groups, each of which will be scrutinized; comparisons will be made when possible. The "old" employees were those employed prior to April 23, 1943.

In order that the appraisal should be fair, it was essential that the orthopaedic surgeon be acquainted with the nature of the work required of the men. Equipped with military passes and accompanied by the proper officials, the author inspected stevedoring operations of all kinds; the physical demands made on the men were appraised, as well as the types of cargo and the methods of handling it.

Cargo brought to the side of the ship by mechanical means must be hoisted or conveyed aboard and then lowered into the hold, either directly or by chutes. Up to this

TABLE I*

	1941	1942	1944	1945
Number of backaches	76	71	37	85
Total number of days lost	908	871	272	188
Average number of days lost per patient	11.95	12.25	7.35	2.2

* Figures supplied by Castle and Cooke Terminals, Ltd.

TABLE II

COMPARISON OF BACKACHES IN GROUPS WITH AND WITHOUT PRE-EMPLOYMENT EXAMINATIONS *

	No. of Men	No. of Backaches	Treatments		Days Lost	
			Total	Average	Total	Average
Stevedores not examined (at least 612)						
First backache	42	42	276	6.6	110	2.6
Subsequent backaches	19	26	86	4.5	47	2.5
Stevedores examined (at least 238)						
First backache	14	14	42	3.0	28	2.0
Subsequent backaches	3	3	7	2.3	3	1.0
	78	85	411	5.3	188	2.4

* Figures supplied by Castle and Cooke Terminals, Ltd.

point, it is mostly machine work. In the hold, every article is moved into its proper place entirely by human strength. The machine operators, hatch tenders, and winch drivers need clear heads; those in the hold, strong backs. It is in the latter group that careful back examinations are required.

Cargo is classified into four main groups: (1) general, (2) special, (3) "icehouse", and (4) bulk cargo. *General cargo* is made up of objects of assorted sizes, shapes, and weights, such as household goods. *Special,* or *repetitive, cargo* consists of objects identical in shape and weight, such as cases of pineapples or sacks of sugar; in handling this cargo, the motions of the stevedores are necessarily repetitious. Work on both of these types of cargo is usually done in hot, stuffy holds. In contrast to these is *"icehouse" cargo,* which is usually of the special type, but is handled at temperatures as low as 10 degrees Fahrenheit. Finally there is *bulk cargo,* such as molasses, oil, grains, and ore. Bulk cargo was not handled by the stevedores discussed here.

With these facts in mind, a scale of employability for stevedores was set up:

Group A: Patients with *good* backs, who exhibited no abnormality by roentgenograms;

Group B: Patients with *fair* backs, who exhibited spina bifida occulta of a single segment, slight asymmetry of the facets, limbus bodies, very early hypertrophic changes, well-reduced healed fractures of the centrum, and old fractures (healed or unhealed) of the transverse processes;

Group C: Those with *poor* backs, who presented marked asymmetry of the facets, sacralized transverse processes, spina bifida involving more than one segment, hemivertebrae, spondylolysis, spondylolisthesis, thin intervertebral discs, Schmorl's nodules, marked hypertrophic changes, and sclerosis of the aorta.

Men in the first two groups were employed; those in Group C were considered not employable.

These standards resulted in the rejection of 51 per cent. of the applicants. However, the man-power shortage was so acute that it was considered necessary to employ some of the better of the poor risks. A fourth group was therefore designated as "questionable risks", in which were included applicants with asymmetry of the facets, severe spina bifida occulta, and moderate hypertrophic changes. The members of this group were then questioned by the Personnel Manager as to a history of backache. If the history was positive, the man was rejected; if negative, he was employed. This method lowered the rejection rate to 30.5 per cent.

RESULTS

The employer is interested in two things: a reduction of the incidence of industrial backache, and a reduced period of disability. 1941 and 1942 were the last two years in

which no back examinations were made. In 1944 and 1945, all stevedores were examined. Each period included one full year and one partial year of war; otherwise the factors were alike. The figures for 1945 are more complete than those for the other years, and reveal several other bits of information. Of the 850 stevedores employed by Castle and Cooke in the latter part of 1945, 612 had been employed without back examinations and 238 had had pre-employment examinations, indicating a wide marginal turnover among stevedores.

The most significant finding in respect to the causes of backache among stevedores lies in the fact that 62 (73 per cent.) of the total of eighty-five backaches in 1945 appeared in the atraumatic group. Of these, forty-four (71 per cent.) developed while the men were working with special, or repetitive, cargo.

Observations concerning treatment and disability are summarized in Table II. The significant findings are the greater number of repeaters in the unexamined group and the greater amount of time lost per backache in this group.

The efficiency of the ratings may perhaps be gauged by the fact that, of the examined group complaining of backache, five had been rated as good and eight as fair. The one poor risk, who had been employed through an error, appears in the backache group.

It would therefore appear that, by pre-employment roentgenographic examinations of stevedores, (1) 30 per cent. of applicants have been eliminated as unfit for this work; (2) the time lost as a result of backaches has dropped by 82 per cent.; and (3) the compensation paid has fallen from about 375 days in the first two years to thirteen days in the last two years.

No objection to this plan of examination has been received from the employees or the labor union; their attitude is rather that of cooperation and appreciation, for they feel that the present system is designed for their protection. This is due largely to the way the problem has been handled by Castle and Cooke's Personnel Department. Each man rejected has had the reason for his rejection discussed with him by a member of the Personnel Department, who has been instructed in the meaning of the various terms appearing in the reports. Each person rejected may consult the author without cost. He is shown his roentgenograms, and the difficulty is explained. A number of persons with correctable conditions have had surgery. All have seemed grateful. One or two with spondylolisthesis, who gave no history of backache, were not reconciled to non-employment as stevedores, because they needed the "big money" ($1.00 per hour).

The medical study should be prefaced by two observations:

1. In a person complaining of backache, an examination during a symptom-free interval is nearly useless.

2. Except in a few conditions, such as fractures and destructive disease of the vertebrae, a roentgenographic examination does not prove that a particular finding is the cause of a specific backache. This is difficult enough with the patient and his roentgenograms lying side by side.

CLINICAL FINDINGS

Concerning these studies, medical men might be interested in several points:

1. Among the private patients, the distribution of the backaches was as follows:

TABLE III

CONGENITAL AND ACQUIRED DEFECTS IN THREE GROUPS OF PATIENTS

	Private Patients (*Per Cent.*)		Old Employees (*Per Cent.*)		Pre-Employment Group (*Per Cent.*)	
No defects	5.3		13.7		18.8	
Congenital anomalies alone	21.9	58.4	13.7	58.7	19.9	51.8
Combined congenital and acquired defects	36.5		45.0		31.9	
Acquired defects alone	36.3	72.8	27.4	72.4	29.3	61.2

TABLE IV

FACTORS CAUSING BACKACHE

	Group Examined Before Employment (*Per Cent.*)	Private Patients (*Per Cent.*)	Old Employees (*Per Cent.*)
Spondylolisthesis	3.8	4.6	2.3
Sacralized transverse process	5.1	8.7	3.8
Thin disc	10.2	25.7	9.9

TABLE V

CONDITIONS WHICH MIGHT PREDISPOSE TO BACKACHE

	Group Examined Before Employment (*Per Cent.*)	Private Patients (*Per Cent.*)	Old Employees (*Per Cent.*)
Asymmetrical facets	26.6	35.0	34.3
Schmorl's nodules	0.95	1.6	2.3
Hypertrophic changes	54.5	61.8	71.0

Neckache occurred in 1.4 per cent. of the patients, thoracic backache in 2.5 per cent., and low backache in 96 per cent. Therefore, a roentgenographic study of the lower part of the back will disclose the potential causes of backache in 96 per cent. of those examined.

2. To determine the relative significance of congenital and acquired defects in the production of backache, each of the three groups—the private patients, the old employees, and the pre-employment group—may be divided into four subclasses, which may then be compared (Table III). From these figures it is apparent that (a) normal backs are fewer in individuals complaining of backache than in an unselected group; (b) employment at heavy labor apparently tends to eliminate individuals with certain congenital defects or causes secondary defects to develop; and (c) acquired defects are of distinctly greater significance in the backache groups than are congenital anomalies.

3. In considering the congenital anomalies which tend to cause an individual to be excluded from heavy labor, it has been assumed that the pre-employment group of 1049 men represent a run-of-the-mill cross section of men engaged in hard labor; that the 560 patients with backache represent a fair sample of individuals with backache; and that the 131 old employees complaining of backache, who were not examined before being employed, would show the eliminative effects of stevedoring on the incidence of back defects. Table IV would seem to point toward three defects—spondylolisthesis, sacralized transverse processes, and thin discs—as the defects most likely to cause disabling backaches among persons performing heavy labor.

4. An effort was made to determine whether there were any defects which, if increased in both groups with backache, would tend to point toward their being predisposing factors to backache (Table V). Heavy labor might either cause the greater incidence of Schmorl's nodules and of hypertrophic changes among the old employees, or it might cause more

TABLE VI

FREQUENCY OF OCCURRENCE

	Group Examined Before Employment (*Per Cent.*)	Private Patients (*Per Cent.*)	Old Employees (*Per Cent.*)
Spondylolysis	4.6	4.1	5.3
Spina bifida occulta	19.0	16.7	27.2
Wedge vertebrae	1.4	1.0	6.9

TABLE VII

HYPERTROPHIC CHANGES

	Group Examined Before Employment (*Per Cent.*)	Private Patients (*Per Cent.*)	Old Employees (*Per Cent.*)
One location	69.0	69.0	56.0
Two locations	24.1	25.5	33.0
Three locations	6.8	5.4	11.0

backaches in persons having these conditions, or both. In any event, the appearance of such conditions should discourage the employment of an individual for heavy labor.

5. Three conditions were studied to see if their frequency of occurrence dropped in the non-stevedore group with backache, but increased in the group doing heavy labor (Table VI). The significance of the observations is not altogether clear. Spina bifida occulta appeared so frequently as a secondary finding among the major causes of backache, listed previously, that its increase was probably of no special significance, but rather something left over after the hard labor had tended to eliminate those individuals having major causes of back disability. The author considers spondylolysis as a potential source of spondylolisthesis, and distinctly a backache hazard, which is brought into prominence by heavy labor.

6. Since hypertrophic changes are likely to occur in numerous places in the same individual, the statistical evidence as to the importance of multiple appearances was considered. Hypertrophic changes have been placed in three anatomical and physiological groups: (a) the longitudinal ligaments, (b) the iliolumbar ligaments, and (c) the sacro-iliac ligaments. Several intervertebral spaces might show changes, but they would all be grouped as one, under longitudinal ligamentous changes. Therefore, we are limited to three possible appearances (Table VII). This table would seem to emphasize the frequency of multiple hypertrophic changes among old employees, and would apparently point toward (a) heavy labor as a cause of the condition and (b) the occurrence of multiple hypertrophic lesions as a cause for the rejection of an individual for heavy labor.

The general group with backache was made up of men and women from nearly all walks of life, but none of the women were stevedores. Table VIII brings out several facts concerning these women: (a) Hypertrophic changes occurred about 50 per cent. more frequently among women than among men; (b) multiple lesions were more frequent in women; and (c) the three areas in which the women showed a more marked predominance of hypertrophic changes than the men were the thoracic, sacro-iliac, and iliolumbar regions. These three areas are the ones most subject to the ever-increasing strains of childbearing. The thoracic area takes the strain throughout the period of gestation, while the pelvic ligaments are more subject to the periodic relaxations of the pelvis and the occasional bursting strains of childbirth. The greater obliquity of the female pelvis may also play a part in this predominance.

TABLE VIII

LOCATIONS OF LESIONS

	470 Males (*Per Cent.*)	90 Females (*Per Cent.*)
Longitudinal ligaments	42.5	44.5
Cervical	1.5	2.2
Thoracic	3.5	8.9
Lumbar	37.5	33.3
Iliolumbar region	9.1	15.5
Sacro-iliac region	24.7	49.8

From these facts we may conclude (a) that the incidence of backache in the sacro-iliac or iliolumbar region is nearly twice as great in women as in men; and (b) that a different set of criteria would be required for prospective women employees.

7. The rarer vertebral anomalies, such as a hemivertebra or a limbus body, have appeared so seldom that it is impossible to draw statistical conclusions. However, judging from limited experience, the author would rate an applicant with a hemivertebra as a poor risk, because this is an asymmetrical development which throws unusual strains on the muscles and ligaments; and, in the older cases seen, the hypertrophic changes have been marked. A limbus body, on the other hand, is a symmetrical development that dates back to amphibian days, where it appeared as the intercentrum in Seymouria. It causes no unusual strains, and hence an applicant with a limbus body would be rated as a good risk. A candidate having a well-reduced and healed fracture of the centrum would be rated as fair, while one with a poorly reduced fracture would receive a poor rating.

8. When rated on the same basis as the pre-employment group, 33 per cent. of the old employees were rated as employable and 67 per cent. as unemployable. Thus the figures for the two groups are nearly reversed. This may mean either that the work is productive of disability or that many persons who were bad risks were employed prior to the institution of the pre-employment roentgenographic examinations of the back. The fact that the so-called old employees averaged about five years younger than the pre-employment group would certainly suggest the latter explanation.

It would appear that the next stage of this work should be the reduction of the type of backache associated with handling repetitive cargo. The following program should be instituted:

(a) Stevedores should alternate the side presented to the stowed cargo every fifteen or thirty minutes, so as to distribute the working load evenly on the two sides of the back.

(b) The stevedores working in hot holds should be supplied with adequate water, and should be compelled to take enteric-coated salt tablets in sufficient amounts to balance the salt loss.

(c) The stevedores should be instructed in the value of resting in dry clothing during the noon hour.

(d) Stevedores working with "icehouse" cargo should have warm drinks or soups available, and should protect their loins with a warm binder. As long ago as 1620, Venner observed that "some soft woolen cloth will preserve from lumbaginous pains".

A system that finds 30 per cent. of thirty-year-old males unfit for heavy labor is freighted with heavy social implications. Some of the men were illiterate and some stupid, but generally they were of average intelligence. Some of the physical defects were beyond remedy, but some of the men could be converted from social liabilities to social assets. The remediable conditions could all have been recognized before the individuals left school; an appraisal of our adolescents would give an opportunity to guide them away from channels for which they are physically unfit.

DISCUSSION

DR. PHILIP LEWIN, CHICAGO, ILLINOIS: I would like to ask two questions: The first is the labor-union aspect, because in Chicago, twenty years or more ago, a group of men (industrial surgeons and roentgenologists) attempted to do this, but the unions prohibited it. Second, what about bilateral sacralization of the fifth lumbar vertebra as compared to unilateral sacralization?

DR. STEELE STEWART (closing): In answer to Dr. Lewin, not only have the men cooperated, but the labor unions have cooperated. This is probably due in a small way to the manner in which the problem was approached and the way it was handled. When a man applies for a job as a stevedore, he is sent to the Medical Group, examined by one man, and a permanent record is made. He is then sent to the x-ray room and three roentgenograms are taken,—one as the back lies on the table, one with the back flat, and a lateral view. The report is written up and sent to the employer, stating the exact condition found in

(*Continued on page 236*)

AN IMPROVED SKIN-TRACTION TECHNIQUE FOR THE FINGERS

BY LIEUTENANT COLONEL HARTWIN A. SCHULZE
Medical Corps, United States Army

The conservative methods of treating oblique fractures of the metacarpals and the proximal phalanges of the fingers ordinarily require prolonged traction. To meet this need, the fingernail method and the soft-tissue transfixion method are usually inadequate. The skin-traction methods which employ various types of adhesive tape or glue are also unsatisfactory, because of early slipping. For these reasons skeletal traction, employing Kirschner wires or tongs, is ordinarily considered the method of choice. The application of skeletal traction, however, has the disadvantages of requiring an expert operator, a precise technique, and special equipment. It is accordingly time-consuming.

Since the skin of the fingers is tough and relatively immobile, skin traction at this site

FIG. 1

Application of bias muslin binder over crinoline gauze.

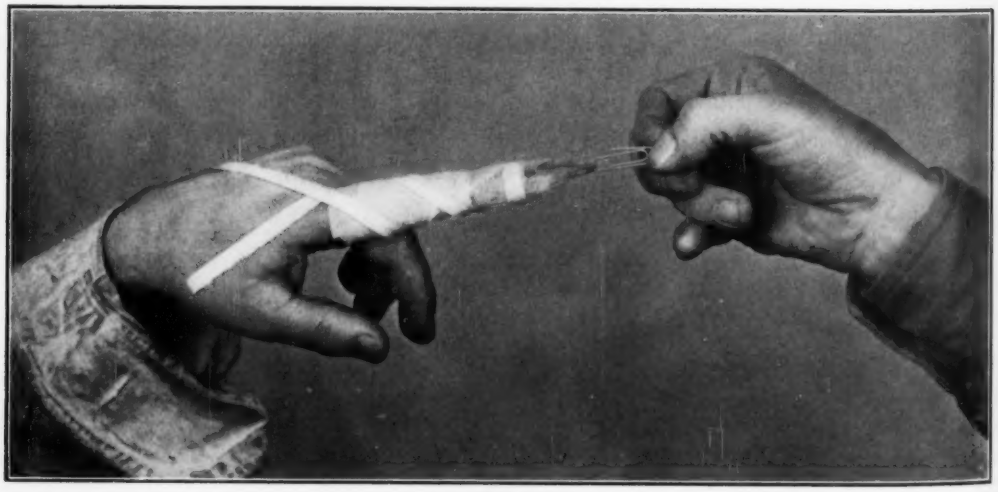

FIG. 2

The completed device, ready for the application of traction.

FIG. 3

Grilled appearance of the skin after removal of the gauze. (×3)

would seem to offer some special advantages, if only slipping could be avoided. Slipping is usually due to a low viscosity of the adhesive substance, and a further reduction of its viscosity by the absorption of the secretions of the skin.

The method described here avoids these difficulties by employing a non-viscid adhesive substance and a bonding technique which depends upon precise coaptation of two irregular, apposing surfaces. Such coaptation is produced by placing a coarse-meshed gauze in contact with the skin, under slight pressure.

The following materials are required:

1. A lacquer with a cellulose base. Most fingernail lacquers are of this type, and there are many lacquers of this kind on the market for general use.

2. A strip of crinoline gauze, having twenty to thirty threads to the inch. This is the starched gauze used in making plaster bandages.*

3. A roll of bias muslin, one-half inch wide. This can be sliced with a knife from an ordinary roll of bias muslin bandage.

The procedure is as follows: The finger is given a liberal coating of lacquer. A strip of the gauze, about one and one-half inches wide and about ten inches long, is applied to the dorsum and the ventrum of the finger, with the loop extending about two inches beyond the tip of the finger. The gauze is bound snugly in place with a strip of half-inch bias muslin, applied in a simple spiral fashion. The muslin binder extends from the base of the finger to the base of the nail and back to the base of the finger (Fig. 1). The end is secured with a bit of adhesive tape. The muslin is kept in place with a narrow strip of adhesive tape, applied as a wide-open spiral. The protruding gauze loop is smeared with lacquer and rolled into a cord. To prevent pressure on the finger tip, a cork disc, cut from a bottle stopper, is used as a spreader. It is placed inside the loop, and held in place with an encircling strip of adhesive tape. Traction can be applied immediately (Fig. 2).

* Surgical gauze of similar mesh, having a hard texture and large threads, may be used, but it is more difficult to handle. Bookbinders' gauze, which is stiffened with glue instead of with starch, is also satisfactory.

When the materials are removed, it will be seen that the skin has a grilled appearance, with the depressions corresponding to the threads of the gauze (Fig. 3). This factor, rather than the adhesive properties of the lacquer, undoubtedly accounts for the greater part of the non-slipping properties of the device. This same principle has a diversity of applications in various fields of engineering.

When this method is used, the only caution necessary is to avoid constriction. If the binder is applied too tightly, it will be apparent in the exposed finger tip within a few minutes. If evidence of constriction is not apparent at that time, it is unlikely to develop. The patient should, however, be instructed to make frequent observations during the first day. The hand should, of course, be carried in a sling.

The only maintenance necessary is to see that the binder does not get too loose. If this happens, a second binder can be applied directly over the first.

This method of traction has the following advantages: It does not slip even when continued over a prolonged period; it can be applied in a few minutes; a technician can readily apply it; and it avoids the risks inherent in skeletal traction. The only disadvantage is that it cannot be used for fractures distal to the proximal phalanx or in the presence of certain concomitant finger injuries.

The author has used this method for many fractures of the metacarpals and proximal phalanges of the fingers without experiencing any failures.

INFANTILE CORTICAL HYPEROSTOSIS

BY DOUGLAS D. DICKSON, M.D., CLARENCE A. LUCKEY, M.D., AND NOBLE H. LOGAN, M.D.,
OAKLAND, CALIFORNIA

Infantile cortical hyperostosis is the descriptive title used by Caffey and Silverman[1] in reporting four cases of a disturbance involving primarily the skeletal system of infants. Their cases were characterized by the sudden onset, within the first three months of life, of tender swelling, the location depending upon the bone or bones affected. Involvement was noted in the tibiae, femora, ribs, humeri, radii, ulnae, clavicles, mandible, and scapula; but not all of these bones were involved in any individual case. There was a striking absence of general systemic manifestations. An elevation of temperature was noted in two of four cases at the onset, and was present later in a third case. Roentgenographic findings revealed cortical thickening and lamination in many instances, with normal-appearing spongiosa, metaphyses, epiphyseal plates, and ossification centers in the epiphyses. Other laboratory studies revealed no significant findings. After many weeks the swelling subsided and no sequelae were noted.

CASE REPORT

S. W. was a full-term female infant, delivered without difficulty or complications on July 12, 1945. At the age of one week, orange juice and cod-liver oil were included in the formula. Nutritional requirements have been met up to the present time. At the age of two months, the mother first noted swelling over the right scapular region. Examination at that time revealed a hard mass over the region of the

right scapula with invasion into the right axilla. There was some duskiness of the skin over this area. The first roentgenograms (Fig. 1) were taken on September 18, 1945. They revealed moderate bony overgrowth of the wing of the scapula, together with thickening of the surrounding soft tissue. Films taken on September 28, 1945, showed an increased reaction in the scapula. Some cortical thickening of the mandible was also demonstrated at that time. Subsequent roentgenograms taken on October 8 and October 15, 1945, showed considerable extension of the process in the scapula and also involvement of the clavicle on the same side (Fig. 2). A biopsy was done on September 28, 1945. There was considerable oedema of the muscles overlying the mass. Microscopic examination revealed invasion of the muscle with inflammatory cells (Figs. 3-A and 3-B).

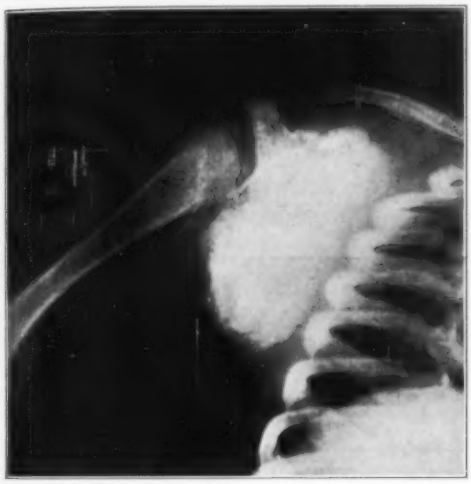

FIG. 1

FIG. 2

Fig. 1: Demonstrating marked bony thickening of the scapula.
Fig. 2: Illustrating further thickening of scapula, and bony proliferation involving the clavicle.

FIG. 3-A

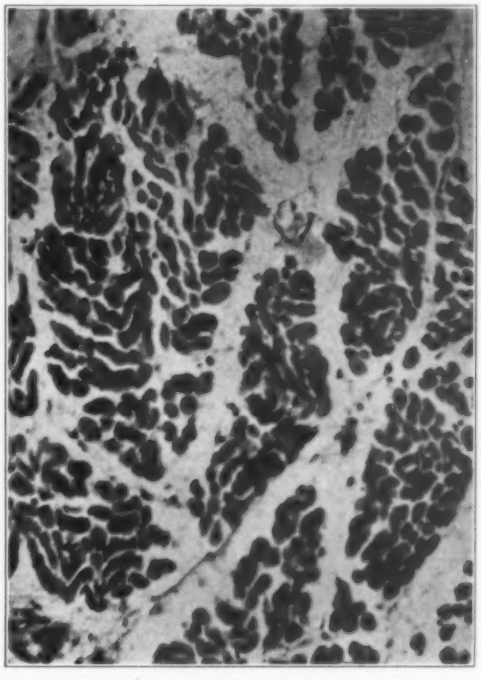

FIG. 3-B

Invasion of the infraspinatus muscle with inflammatory cells.

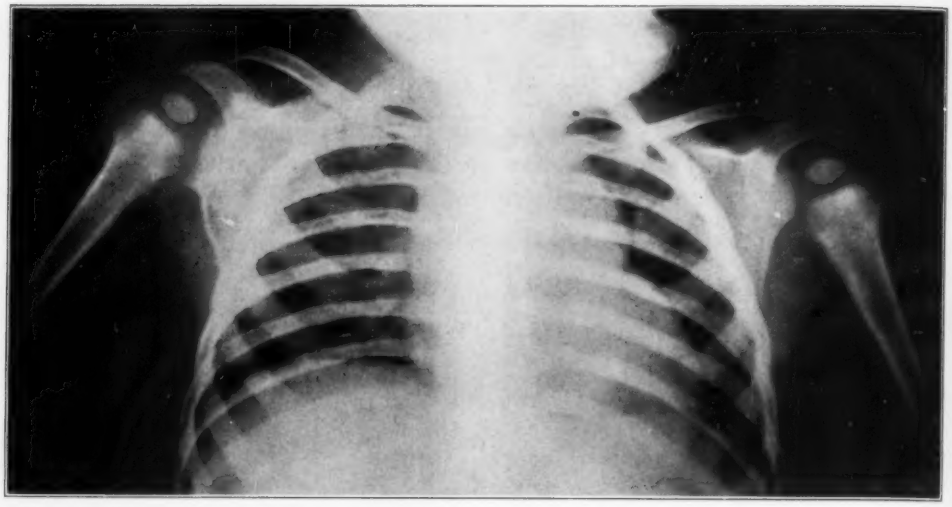

FIG. 4

Showing restoration of normal bone pattern in both clavicle and scapula.

Then gradual regression of the mass began. Roentgenograms taken on November 11, 1945, showed that the bony proliferation in the right scapular region had subsided considerably. On April 5, 1946, the roentgenograms showed only slight residual thickening of the right scapula. Final roentgenograms on April 26, 1946, revealed a normal bone pattern throughout the entire skeletal system (Fig. 4). The Kahn and Kline tests and the Wassermann reaction were negative.

During the hospital stay, there was elevation of temperature to 100.6 degrees by rectum on one occasion; usually the temperature was not over 99.5 degrees by rectum. There was adequate vitamin intake previous to, and during, the hospital stay.

TABLE I

HEMATOLOGICAL FINDINGS

Date	ANALYSIS			DIFFERENTIAL COUNT			
	R.B.C.	Hemoglobin	W.B.C.	Neutrophils	Lymphocytes	Monocytes	Eosinophils
Sept. 29	3,040,000	8.2 grams	17,450	71	21	6	2
Oct. 12	3,140,000	7.9 grams	14,550	37	57	6	
Oct. 18*	6,550,000	23.4 grams					

* Following transfusion.

The etiology is obscure. Whether or not this is a response to infection, cannot be settled easily. The biopsy does not suggest that osteomyelitis is the cause; however, infants may respond differently than adults in the presence of a hematogenous osseous inflammatory lesion. It has been noted frequently that osteomyelitis in the infant is benign and produces no sequestra.

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OSTEOMYELITIS CAUSED BY *SALMONELLA TYPHIMURIUM*

BY RUTH F. KRAUSS, M.D., BUFFALO, NEW YORK

From the Children's Hospital and the University of Buffalo School of Medicine, Buffalo

It is generally recognized that osteomyelitis in man may be caused by a variety of micro-organisms. Most commonly encountered are the Staphylococcus and beta-hemolytic Streptococcus, and less often the pneumococcus, influenza bacillus, typhoid bacillus, and others. That Salmonella organisms only rarely cause an infection of bones and joints is evidenced by the fact that, even though the first of such cases was described as early as 1896, a review of the literature in 1940 by the Uruguayan investigators, Guerra, Peluffo, and Aleppo, disclosed but fifteen authentic cases. *Salmonella suipestifer (Salmonella choleraesuis)* was the causative agent in twelve of the fifteen cases reviewed by Guerra and his associates. In the remaining three cases, *Salmonella typhimurium* was the incitant in two instances and *Salmonella enteritidis* in one. Other Salmonella types have been recovered from osteomyelitic lesions,—for example, *Salmonella panama*[1] and *Salmonella schottmülleri*[2]. A search of the literature failed to disclose a report of osteomyelitis due to *Salmonella typhimurium* in which a large number of bones were involved. The pertinent clinical, bacteriological, immunological, and roentgenographic aspects of such a case are, therefore, presented here.

CASE REPORT

F. J., a white boy, two and one-half years old, was admitted to the Children's Hospital on September 2, 1945. The family history and the past history were non-contributory. The present illness began on September 1, with fever and listlessness. On admission to the Hospital, the boy's temperature was 106.4 degrees, the pulse rate was 150, and the respiratory rate was 35 per minute. The patient was well developed and well nourished, but appeared pale, listless, and acutely ill. His neck was stiff. Examination of the chest revealed diminished resonance and tubular respiration on the left side. No other significant changes were noted.

Clinical Course

During the first fortnight, the patient's general condition remained serious. The temperature fluctuated between 102 and 105 degrees during the first week, and then ranged from 99 to 105 degrees during the following week. Roentgenographic examination of the chest showed small amounts of fluid in both pleural cavities. On September 15, aspiration yielded eighty-five cubic centimeters of straw-colored fluid from the left pleural cavity; this fluid proved to be sterile on culture. During the next two weeks the general condition of the patient improved, and the temperature reached lower levels (99 to 103 degrees). Early in October, about one month after admission, tenderness over the ribs and the right scapula was noted. Roentgenographic examination disclosed for the first time widespread osteomyelitic lesions of almost all the ribs and of the right scapula (Fig. 1). The wings of the ilia also were involved, but all other bones appeared to be normal. On October 20, new osteomyelitic lesions were discovered roentgenographically in the neck of the left femur and in the portion of the right ilium adjacent to the acetabulum. Figure 2 is a reproduction of the roentgenogram of the chest, taken on that day. The patient's general condition showed continued improvement, and the temperature became normal two months after admission to the Hospital.

On November 6, roentgenographic studies showed marked improvement of the lesions of the ribs, right scapula, wings of the ilia, and neck of the left femur. Figure 3 is a reproduction of the roentgenogram taken then, showing involvement of both ilia, the left hip, and the right acetabulum. Medial displacement of the right ischium and pubis indicated involvement of the cartilage. On November 21, when the patient was re-examined, the ribs were almost normal in appearance; however, there was no change in the dislocation of the ischium and pubis.

The child was examined again on March 21, 1946, approximately six and one-half months after the onset of illness, and he appeared clinically well. The roentgenographic appearance of the ribs and right scapula was normal (Fig. 4) and only a slight residual deformity was present in the wings of the ilia and in the right acetabulum (Fig. 5).

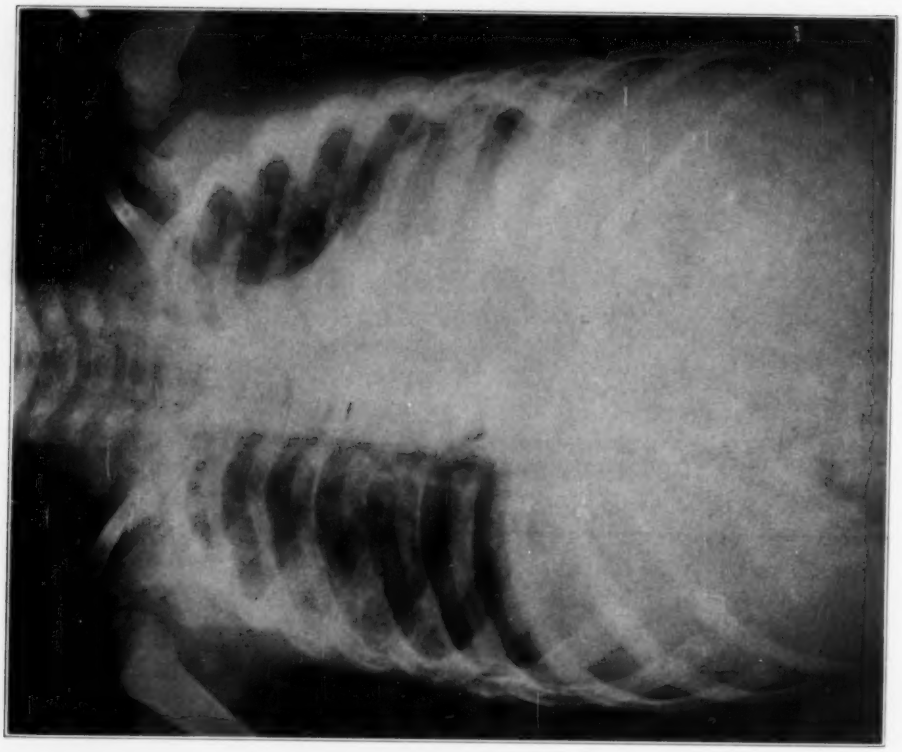

FIG. 2
Roentgenogram of chest, taken on October 20, 1945.

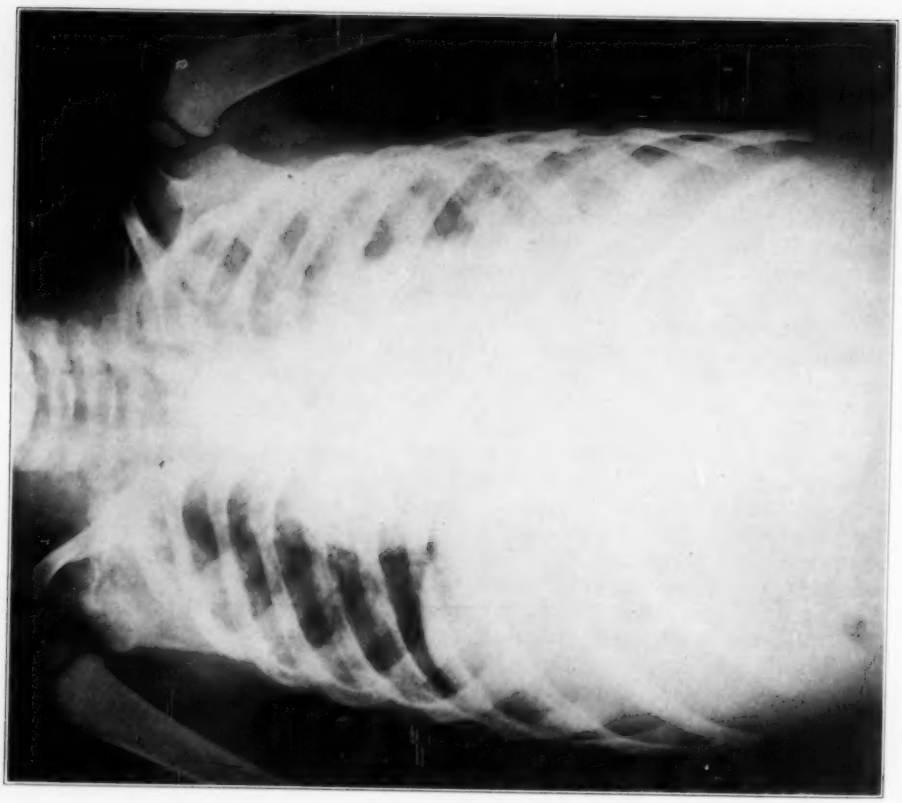

FIG. 1
Shows osteomyelitic lesions of almost all ribs and of right scapula.

FIG. 2
Roentgenogram of chest, taken on October 20, 1945. Shows osteomyelitic lesions of almost all ribs and of right scapula.

FIG. 3
On November 6, there was involvement of both ilia, the left hip, and the right acetabulum.

Therapy

The patient was given one grain of sulfadiazine per pound of body weight (thirty grains) by mouth each twenty-four hours during the first five days in the Hospital. This treatment was resumed on the seventh hospital day, and continued for one week. The resulting blood levels ranged between three and six milligrams per 100 cubic centimeters. From September 17 to December 10, the dosage of sulfadiazine was doubled (sixty grains in twenty-four hours); this resulted in concentrations of the drug in the blood ranging from seven to twelve milligrams per 100 cubic centimeters.

During the first eleven days in the Hospital, the patient was also given from 120,000 to 150,000 units of sodium penicillin intramuscularly each day, in eight divided doses, although it was appreciated that this drug is largely ineffective against Salmonella organisms. During the first ten critical days, the child was given oxygen. Eight blood transfusions, varying in amounts from 80 to 150 cubic centimeters, were given during the first two weeks.

Laboratory Findings

Blood Cultures: A blood culture, taken on September 2, contained gram-negative bacilli with the morphological, cultural, and antigenic characteristics of *Salmonella typhimurium*. The number of organisms per cubic centimeter of blood was too large to be determined. A blood specimen, taken on September 5, was also positive for *Salmonella typhimurium*. On September 14, fifty-two colonies of *Salmonella typhimurium* per cubic centimeter of blood were present. On September 19, the number of Salmonella organisms was greatly reduced, since they were recovered only from the broth culture and not from the blood agar. Subsequent blood cultures, taken on September 25 and October 1, 1945, remained sterile. These data, therefore, indicate that the patient had been suffering from *Salmonella typhimurium* bacteriaemia over a period of at least two and one-half weeks.

Biopsy Culture: Culture of the material obtained by biopsy from the ilium showed the presence of *Salmonella typhimurium*. The histological examination of the biopsy specimen by K. L. Terplan, M.D., revealed the presence of very extensive osteitis and osteomyelitis. The bone marrow was in a fibrous state in some areas; in other parts, granulomatous lesions were noted. A large number of leukocytes

Fig. 5

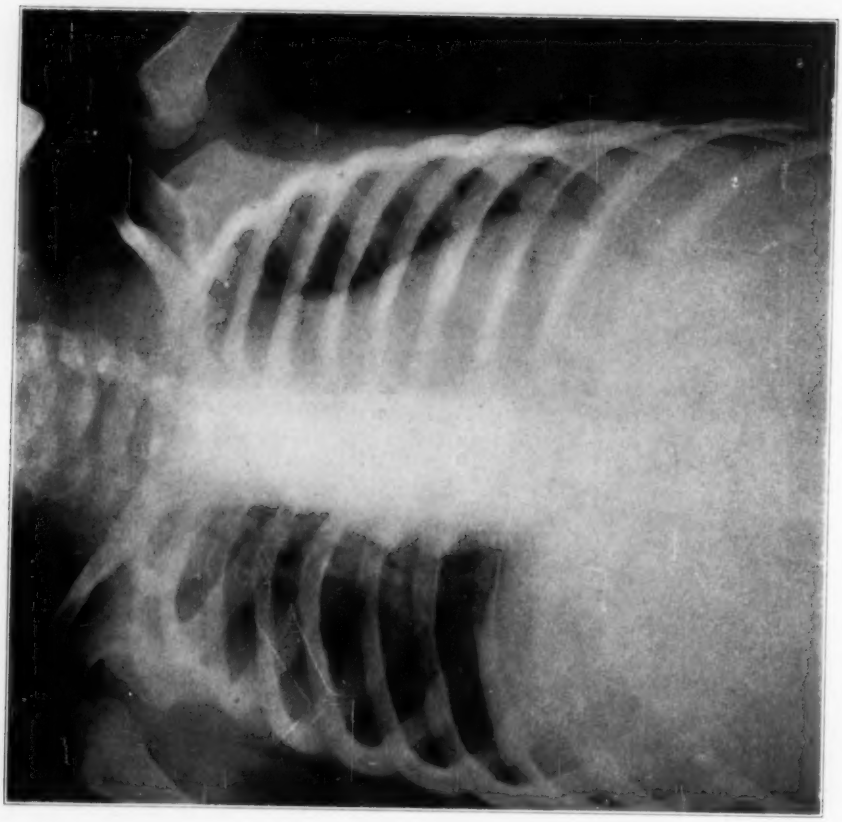

Fig. 4

Roentgenograms taken on March 21, 1946.

were present. It is evident, therefore, that the lesion in the bone marrow was inflammatory in nature and due to an infection by *Salmonella typhimurium*.

Other Cultures: Cultures of faeces and urine and of throat and pharyngeal secretions failed to disclose the presence of *Salmonella typhimurium*, although a special search for this organism was undertaken and repeated examinations were made. The spinal and pleural fluids, likewise, did not harbor the Salmonella organism and proved to be sterile on culture.

Widal Test: The antibody response of the patient was studied by means of the Widal test. A serum specimen, obtained on September 8, showed no agglutinins in titers of 1 to 20 and above, against typhoid, paratyphoid A, paratyphoid B, Proteus, and Brucella organisms. On September 17, agglutinins could be demonstrated; strong agglutination was obtained with a suspension of paratyphoid B. The titer of agglutinins of the serum against the patient's own strain was 1 to 400, and this was unchanged on September 19. On October 29, the agglutinin titer had increased to 1 to 800. It is evident, therefore, that during the course of the illness specific antibodies were developed in a fairly high titer.

Blood Examination: On September 2, 1945, the following blood findings were obtained:

Red blood cells	2,130,000
Hemoglobin	6 grams per 100 cubic centimeters
White blood cells	6,700
Polymorphonuclear neutrophils	
Adult forms	56 per cent.
Young forms	20 per cent.
Lymphocytes	22 per cent.
Monocytes	2 per cent.

During the course of the illness the anaemia slowly disappeared, and the hemoglobin reached a level of 14.5 grams by the time of discharge. The leukocyte count increased to 10,700 on September 6 and reached a maximum of 19,950 on September 19. It became normal on October 16 and remained so until the time of discharge.

Urine Examination: During the entire period of hospitalization, numerous urine specimens were negative for albumin, sugar, and cellular elements.

In summary, then, it may be stated that this child, two and one-half years old, was suffering from septicaemia, caused by *Salmonella typhimurium* and complicated by osteomyelitis of most of the ribs, the scapula, pelvic bones, and femur. Complete recovery took place. It is noteworthy that there was no sequestration in any of the lesions, and that the architecture of the bones returned to normal much more rapidly and completely than in the ordinary case of osteomyelitis caused by the Staphylococcus. Leukopenia was present at the onset of the illness. Agglutinins developed in fairly high titer (1 to 800) against the offending organism. Salmonella organisms were not recovered from the faeces, urine, or pharyngeal secretions.

DISCUSSION

In human beings, Salmonella organisms may produce three different clinical syndromes,—namely, (1) Salmonella fever, resembling typhoid fever; (2) Salmonella gastroenteritis, or food poisoning, and enterocolitis; and (3) Salmonella septicaemia. In Salmonella septicaemia, metastatic lesions may develop in various organs of the body. According to the literature, hematogenous osteomyelitis and arthritis are of relatively rare occurrence. In a review of the reports on Salmonella infections of bones and joints published up to 1940, Guerra, Peluffo, and Aleppo collected only fifteen proved cases. Several other cases have been described or mentioned briefly in various communications, but are not included in the review of Guerra and his associates. For example, in 1925, Ssokoloff described surgical complications of paratyphoid Erzinjan infections; and reported twelve instances of osteomyelitis. Leveuf and his associates refer to seven such cases, reported in the French literature between 1913 and 1928. Still other cases may have been observed but not reported. In view of the repeated finding that Salmonella osteomyelitis often is a relatively mild disease that may improve without surgical intervention, it seems fair to assume that such cases may have escaped diagnosis. In the future, it may be wise to examine roentgenographically the skeletons of patients suffering from Salmonella septicaemia, in order to determine the true incidence of Salmonella osteomyelitis.

Since many of the 150 or more types of Salmonella organisms may cause disease in man, it is not surprising to find various types also as causative agents of osteomyelitis. Much of the older literature on Salmonella infections is of limited value today, in so far as type diagnosis is concerned. For example, in the light of present-day knowledge of the antigenic structure of Salmonella organisms, the paratyphoid Erzinjan bacillus isolated by Ssokoloff may have been *Salmonella enteritidis* (variety from Moscow).

In the present case, the diagnosis of the osteomyelitic lesions in the various bones was established both by clinical and roentgenographic examinations. That the osteomyelitis was due to an actual infection by the Salmonella organism was proved by microscopic and bacteriological examination of the material removed at operation from the involved right ilium. Microscopically, gram-negative bacilli were seen; the culture produced a pure growth of *Salmonella typhimurium*. The biopsy specimen was obtained at a time when the blood culture had become sterile. Consequently, the presence of these micro-organisms was not due to contaminating blood. It seems fair to assume that the osteomyelitic lesions in the other bones were due likewise to actual infection.

Complete recovery took place in the present case. It is impossible to appraise the role played by the sulfonamides and penicillin in the favorable outcome.

NOTE: The author wishes to thank Erwin Neter, M.D., and G. Newton Scatchard, M.D., for the bacteriological and roentgenographic data.

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SALMONELLA INFECTION INVOLVING THE KNEE JOINT

REPORT OF A CASE*

BY THEODORE H. VINKE, M.D., AND HAROLD F. DOWNING, M.D., CINCINNATI, OHIO

A six-months-old white infant was admitted to the Children's Hospital, Cincinnati, on April 7, 1945. A history was obtained that, on April 1, the child had been cross and irritable and his right knee gradually became stiff. The child held the knee in a flexed position and refused to extend it. These symptoms persisted until the time of the first examination.

One month prior to the onset of symptoms, the child had diarrhoea, lasting two weeks. He had had no other illnesses, and the family history was not contributory.

On physical examination, the child appeared well developed and well nourished. He did not appear to be acutely ill, although he was irritable. The skin was dry and no eruption was present. The eyes, ears, nose, and throat were normal. There were no palpable lymph nodes. The lungs were normal and clear to percussion and auscultation. The heart was not enlarged; the rhythm was regular; and the sounds were of good quality. The abdomen had a convex contour. There was no tenderness or rigidity.

* Read before the Clinical Orthopaedic Society, Cincinnati, Ohio, October 1945.

There were no palpable organs or masses. The right knee was held in 90 degrees of flexion, and was mildly tender to anterior pressure over and around the patella. Extension of the knee produced considerable pain. No local heat or discoloration was present. The circumference of the right knee was twenty-four centimeters and that of the left knee was twenty-two centimeters. A definite, marked effusion was present in the right knee joint. The skin was slightly excoriated,—probably the result of heat applied over the joint by the parents; the extremities were otherwise normal. The reflexes were physiological. No paralysis or anaesthesia could be demonstrated. The cranial nerves were intact.

Roentgenograms showed no pathological changes of the bones, but soft-tissue swelling was noted. The laboratory findings were as follows:

April 7, 1945	
Red blood cells	4,300,000
Hemoglobin	11.4 grams
White blood cells	10,050
Polymorphonuclear neutrophils	53 per cent.
Monocytes	5 per cent.
Lymphocytes	41 per cent.
Eosinophils	1 per cent.

A tuberculin test, in which 0.1 cubic centimeter of a purified protein derivative was employed, was negative.

April 14, 1945	
White blood cells	19,050
Polymorphonuclear neutrophils	61 per cent.
Lymphocytes	39 per cent.
Blood Kahn test	Negative
Blood culture	Negative
May 2, 1945	
Red blood cells	4,250,000
Hemoglobin	9.7 grams
White blood cells	8,250
Polymorphonuclear neutrophils	27 per cent.
Lymphocytes	72 per cent.
Monocytes	1 per cent.
Urinalysis	
Appearance	Clear
pH	6.0
Specific gravity	1.013
Albumin and sugar	0
Microscopic appearance	Normal

On May 3 and 4, stool examinations were negative for Salmonella organisms. Salmonella species (Montevideo type) grew in the fluid which had been aspirated from the knee. Blood agglutination against the organism found in the knee fluid was complete in dilutions of 1 to 80 and 1 to 160; it was partial in dilutions of 1 to 20, 1 to 40, 1 to 320, and 1 to 640.

Course: During the first week, the temperature remained at about 100 degrees. It rose to 104 degrees at the end of the week, remained high for three days, and fell to normal by lysis in the next two weeks. The fluid in the knee joint was aspirated on April 21, and again on April 24. About twenty-five cubic centimeters of amber-colored fluid was obtained on each occasion. Penicillin was started on April 10, before the exact diagnosis was known, and given in a dosage of 10,000 units every three hours until April 24. On April 24, sulfadiazine was given, because the organism proved slightly sensitive to this drug in the laboratory. The child was given thirty grains of sulfadiazine daily until May 5. The knee became less swollen, and the range of motion gradually increased until normal motion was restored. In all probability, the drug therapy did not influence the recovery.

The organism was a non-spore-forming, gram-negative rod, identified as Salmonella species (Montevideo type) by its fermentative reaction on the different carbohydrates.

There have been seventeen known outbreaks of Salmonella infection of the Montevideo type in the United States, but no other report has been found of knee-joint involvement.

UNUNITED EPIPHYSIS OF THE ISCHIUM

Report of a Case

BY HARRY WINKLER, M.D., AND IRA H. RAPP, M.D., CHARLOTTE, NORTH CAROLINA

During recent years, interest has been directed to a rather infrequent lesion of the tuberosity of the ischium, designated variously as "avulsion fracture of the ischial tuberosity", "isolated fracture of the ischium", "hamstring avulsion", "epiphysitis of the tuberosity of the ischium". Fractures of the ischium are usually associated with multiple fractures of the pelvis, and are commonly the result of direct violence. Heretofore, fracture of the ischium, due to indirect violence, was thought to be exceedingly rare. Cotton failed to report a single case, although he recognized the possibility of its occurrence. Key and Conwell state: "Fracture of the tuberosity of the ischium is a very rare injury". Watson-Jones records the fracture as "hamstring avulsion". The first reported case in the American literature was that of Berry's in 1912, and since that time other authors have reported its occurrence[1,3,7,8,9].

The mechanism of fracture, or, more properly, epiphyseal avulsion, is a violent force, transmitted indirectly by contraction of the hamstring muscles to the secondary epiphysis of the tuberosity of the ischium. The ischium is ossified through two centers, one primary and one secondary. The secondary center for the tuberosity does not appear until puberty, and is not completely united to the body until the age of twenty-five[5]. It is, therefore, a fracture of early life. The displacement of the fragment is determined by a resultant force, which in turn is the product of multiple component forces: the hamstrings and adductor magnus pulling downward, outward, and forward; the quadratus femoris and gemellus inferior pulling upward, outward, and forward; and the sacrotuberous ligament, the direct antagonistic force, preventing a thrust downward, outward, and forward.

The history is highly suggestive of the diagnosis; the injury is usually sustained while the person is running and when the hamstrings are in the active phase of contraction, although in Milch's case the patient was doing the "split" in a fancy dance. There is sudden and severe pain in the involved buttock, and walking becomes painful or impossible. The patient subsequently improves on conservative therapy, although he may be subject to minor exacerbations of pain with strenuous exercise or prolonged weight-bearing. The course of events then may be toward complete symptomatic improvement; or the detached epiphysis may become enlarged, necessitating operative removal. With the exception of the case reported by Berry, surgical removal of the lesion has not been undertaken.

This case is presented because of the persistence and progression of symptoms which necessitated the removal of the fragment, and afforded an opportunity for study of the tissue histologically.

P. S., a man, aged thirty-nine, stated that, at the age of fourteen, he had been participating in a broad jump. While running and preparing for the leap, he felt something "snap" and immediately he experienced severe pain in his left buttock, which radiated along the posterior aspect of the thigh. He fell and had to be carried from the field of play. The family physician advised bed rest, and after several days of rest he returned to school. Later he was able to participate actively in baseball, but experienced minor exacerbations of pain in the buttock and weakness of the left leg.

In January 1946, because of persistent and increasing pain, especially when sitting, he sought medical aid. On clinical examination, the patient was observed to be a generally well-developed and well-nourished white male. No abnormality of gait was noted, and his posture was not remarkable. The lengths of the legs were equal, and the muscle power in both lower extremities was rated from good to normal. There was no change in reflexes of the lower extremities. Local examination disclosed a firm, smooth, non-movable mass posteriorly, in the region of the left buttock; otherwise the general physical examination was essentially negative. A roentgenogram taken at that time revealed a large detached

FIG. 2
Roentgenogram taken February 20, 1946, showing the ischium after the removal of the fragment.

FIG. 1
Roentgenogram, taken on January 21, 1946, showing the ununited epiphysis of the left ischium.

crescent-shaped fragment in the region of the left ischial tuberosity. On February 2 the mass was excised by the senior author. With the patient in the lithotomy position, a longitudinal incision was made from the superior margin of the pubis on the left, extending in a downward and parallel direction to the inferior pubic ramus. On deeper dissection, a firm mass was encountered, which was roughly ovoid in shape and approximately three inches in length. The line of cleavage between the normal bone of the ischium and that of the tumor was identified, and the mass was removed by dissection in this plane. This tissue, with shavings from the line of attachment, was sent for pathological examination.

The pathological report revealed an ovoid mass, measuring 7 by 4.5 by 4 centimeters. Histologically, the tissue resembled normal osseous matter with normal marrow elements, the exterior being partially lined by hyaline cartilage and dense tendinous connective tissue. Microscopic sections of the shavings from the line of attachment of the mass showed resting hyaline cartilage, with a paucity of nuclei and with areas of transition into dense connective tissue of the tendinous type.

The patient made an uneventful recovery, and six months later is asymptomatic with no weakness of the left leg; he can now sit comfortably for any length of time.

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Steele F. Stewart (*Continued from page 221*)

the back. The man is brought in and the statement is read to him by a member of the Personnel Department. The staff of that Department have been instructed as well as they can be that, when a serious condition appears in the report, they should try to explain it to the man so that he will understand it. If he cannot understand it, he has the privilege of coming to see me without cost; I will go over the roentgenograms and explain the condition to him. In other words, we have a very high degree of personal relationship with the individuals. I think the closer we can come to personal relationships in our labor dealings, the less trouble we are going to have with labor unions in this sort of thing.

Regarding bilateral sacralization, I do not believe this is as dangerous a risk as unilateral sacralization. We must remember that, in bilateral sacralization of the transverse process, we are likely to have a thin disc, and that a thin disc is anatomical and physiological,—not an abnormal disc. That type would not fall into the group we have mentioned.

OSSIFICATIONS ASSOCIATED WITH CHRONIC STRAIN OF THE TIBIAL COLLATERAL LIGAMENT FROM ROLLER-SKATING

BY EDMUND W. KLINEFELTER, M.D., YORK, PENNSYLVANIA

Although roller-skating is a very popular sport and injuries resulting from it are frequent, practically nothing concerning them has appeared in the medical literature.[2] Over the past thirteen years, many patients with these injuries have been referred to the author for examination and treatment. The most frequent and most interesting injuries have been acute and chronic strains of the tibial collateral ligament of the knee, associated with ossifications.

Chronic strains of the upper half of the tibial collateral ligament, which often occur among football players and skiers[3], do not result from the usual skating movements, but are produced by such movements as the "split" in figure skating. Subsequent to these strains, ossifications may develop in this part of the ligament (Fig. 1). Similar ossifications, resulting from causes other than roller-skating, have received considerable discussion under the heading of Pellegrini-Stieda disease[1] (Fig. 2). They have, in principle, the same significance and require the same treatment as those in the lower half of the ligament.

Since the stress in roller-skating is applied more to the lower half of the ligament, strains resulting in ossifications are more frequent in this portion than in the upper half. In addition, ossifications appear also at the strained tendon attachments of the popliteus and soleus muscles, on the medial aspect of the tibia. These ossifications frequently produce symptoms and may interfere seriously with the function of the attached ligament and tendons. Therefore, they are important; but no report concerning them could be found.

Because of the stress from an added load and ill-adapted movements, these chronic strains with ossifications are especially predisposed to develop in the obese woman with a square torso, protruding abdomen, and relatively long, thick legs, as she advances beyond the age of thirty-five years. This woman usually lacks ease and grace on the crowded rink. In order to avoid collisions with other skaters, she is compelled to check her advance frequently, not by the turning and gliding movements characteristic of the younger, better-developed skater, but by throwing her weight on the inside wheels of the externally rotated skate as it is dragged against the floor; the same leg is practically always used for this purpose. Tremendous stress is thereby placed on the tibial attachments of the tibial collateral ligament, and of the popliteus and soleus muscles.

The development of the strain is gradual; the first symptoms rarely appear before at least three years of skating. Early in the development of the strain, the skater may notice slight pain in the knee, with perhaps a slight feeling of rubbing, and at times audible creaking on movement. The day after a period of skating the knee may feel somewhat stiff, but this stiffness tends to disappear. Physical examination may disclose slight tenderness over the tibial attachments of the tibial collateral ligament and the popliteus and soleus muscles, slight limitation of flexion, and pain localized to the ligament and tendon attachments upon passive abduction of the leg at the knee. A roentgenogram may show the swollen ligament (Fig. 3). After several days of rest, all symptoms usually disappear.

Frequently the skater herself discovers that the checking movements cause trouble, and she avoids them. In any case, the importance of avoiding these movements must be impressed upon her. Moreover, it is well to recommend a shorter, slower skating stroke, and limitation of the total skating period to not over one hour per week. If these recommendations are not observed, bony deposits, visible on the roentgenograms, often begin to appear at the region of the tibial attachments of the tibial collateral ligament and of the popliteus and soleus muscles (Fig. 4).

These deposits represent an additional warning sign against a continuation of the

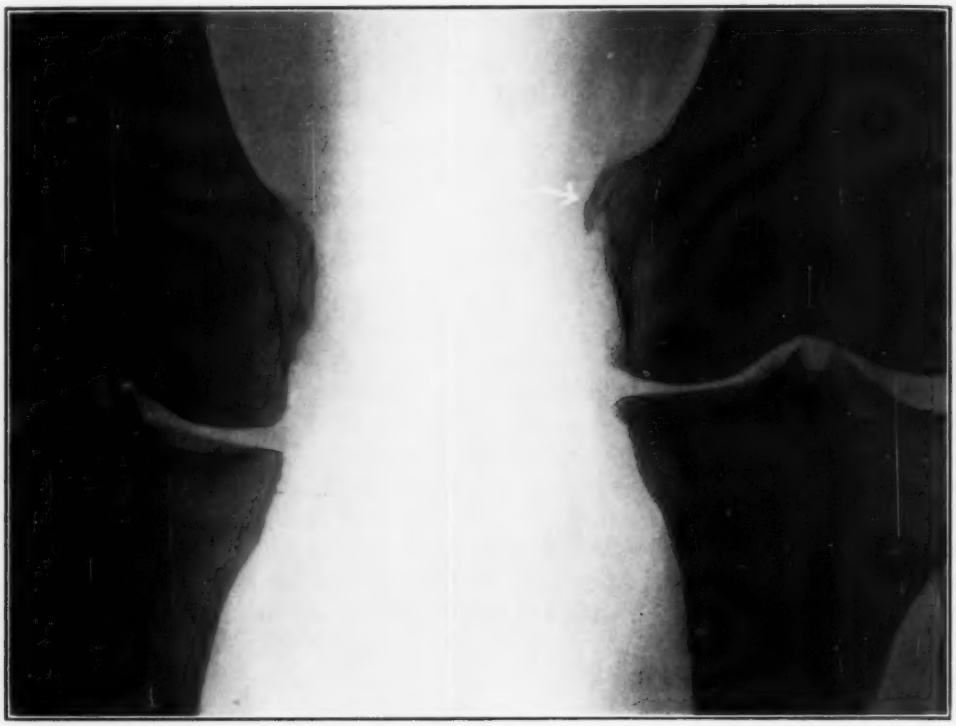

FIG. 1

G. C., a woman, aged fifty-three years, had chronic strain of the right tibial collateral ligament, with an osseous deposit at the femoral attachment of the ligament and advanced degenerative changes of the medial aspect of the right knee joint, resulting from thirty-one years of figure skating. (Illustration reversed.)

FIG. 2

I. C., a woman, aged forty-two years, had chronic strain of the right tibial collateral ligament with an osseous deposit in the ligament (Pellegrini-Stieda disease), resulting from twenty-seven years of figure skating.

FIG. 3

D. L., a woman, aged forty-eight years, had chronic strain of the left tibial collateral ligament. Roentgenogram shows the swollen ligament, resulting from five years of frequent checking movements. Osseous deposits on the medial side of the right knee resulted from twenty-eight years of figure skating.

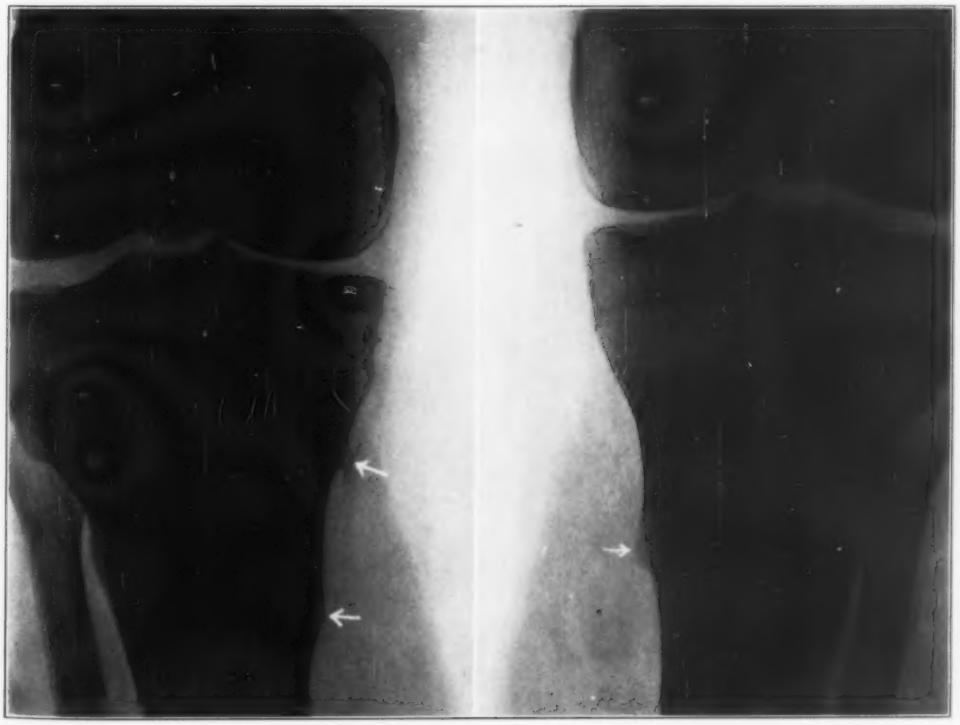

FIG. 4

D. T., a woman, aged forty-four years, had chronic strain of the right tibial collateral ligament and the tendons of the popliteus and soleus muscles, with osseous deposits at their medial tibial attachments, resulting from twelve years of frequent checking movements in roller-skating.

sport in strenuous form. As long as the stress is continued, the ossifications become progressively larger, with the result that the attached ligaments and tendons are strained more easily and more severely, and the strains remain longer. With discontinuance of stress, the strains gradually disappear; but the ossifications usually remain,—ready to cause trouble if stress is reapplied.

When these ossifications are present, a short period of skating and the use of checking movements may cause such severe pain that the skater is unable to continue, and is often compelled to hold the knee as if paralyzed. If the stress is avoided for several days, the severe pain and disability tend to disappear, leaving a residual soreness and stiffness which may persist for several months. For this condition, roentgenotherapy has been found most effective.

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AN EFFICIENT DUAL-PURPOSE RETRACTOR

BY LIEUTENANT COLONEL PETER-CYRUS RIZZO
Medical Corps, Army of the United States

Proper tissue retraction is essential to successful and expeditious surgery. Most retractors are mechanically unsound, as a shift of position tends to disengage them.

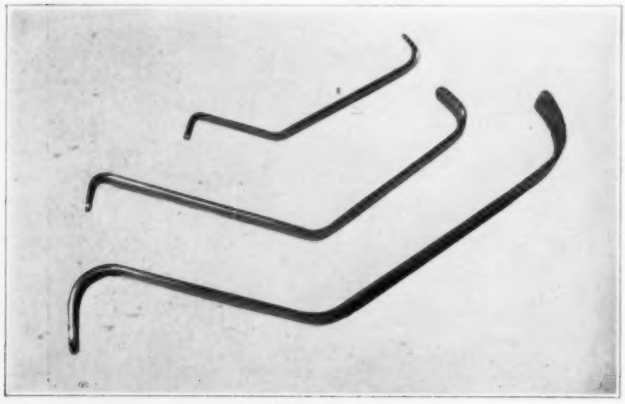

Fig. 1

The principle of the retractor described here lies essentially in the 45-degree angle of the handle and in the shaping of the handle end. The angulation allows transmission of force and pull to the tip, or toe, of the retractor. The ordinary retractor transmits the pull to the heel, and thereby predisposes to slipping and loss of effect. The handle ends are so shaped (Fig. 1) that they can be used for retraction or for isolation of bone or soft tissue. The angle is placed approximately four inches from the heel in the large retractor and one and one-half inches from the heel in the small retractor. The smallest size is most useful in surgery of the hand or of the small parts; the largest size has no limitations, depending upon the length of the foot, or claw end.

A MINIATURE GALVANIC STIMULATOR

BY JAMES E. BATEMAN, M.D., TORONTO, ONTARIO, CANADA

From the Division of Orthopaedic Surgery, Christie Street Hospital, Toronto

The value of electrical stimulation in nerve injuries has frequently been questioned. However, during the recent War, such reports as those by Solandt and associates, and Gutmann and Guttmann have appeared as strong recommendations.

From these experimental and clinical investigations, it may be concluded that definite changes occur following electrical stimulation of denervated muscle. Histologically, it is evident that, by minimizing atrophy, the serious damage at the motor-end plate is decreased. It has become apparent that diminished atrophy and all its associated benefits may be expected only when treatment is applied early, at frequent intervals, and for a prolonged period. Electrical stimulation will not entirely prevent damage in denervated muscle; but, if it is instituted early and given frequently, the muscle will be preserved in as near to the ideal condition as possible.

The application of the principle of adequate stimulation in a large group of patients with nerve lesions has proved difficult, chiefly because of limitations of staff, space, and equipment. The accommodation alone of such patients, even in a large center, is difficult, and attendance at the Physiotherapy Department becomes unsatisfactory. This crowding and confusion has resulted in patients receiving a short period of treatment once every two or three days. Irregular and infrequent stimulation cannot be expected to minimize the damage of denervation significantly. Moreover, one is reluctant to keep a patient under full hospital surveillance for many months, in order to give treatment lasting only a few minutes each day. The earlier the patient can be returned to his occupation, the better. Also, despite the merits of the case, commercial firms and employers are reluctant to allow a man an hour a day from his job for several months. If the patient can carry on at his job, receiving adequate therapy without time loss, rehabilitation is accelerated. To meet such difficulties and at the same time to increase the amount of therapy, experiments were undertaken to produce a small pocket-size stimulator which the patient might wear and use himself, reporting at frequent intervals for supervision.

FIG. 1

The pocket-size electrical unit. The controls are all on one panel to make possible operation with one hand.

THE TECHNIQUE OF STIMULATION

Galvanic current has proved the most satisfactory and practical means of stimulating denervated muscle. Sinusoidal or surging current is excellent, but not all patients respond to such stimulation. For maximum results from galvanic stimulation, small electrodes should be placed on either end of the muscle, and each electrode shifted when a new muscle is to be stimulated. While such a technique has always been prescribed, investigation has shown that it is rarely carried out carefully. Invariably, treatment consists in using a large indifferent electrode with a small point stimulator to pick out the individual muscles.

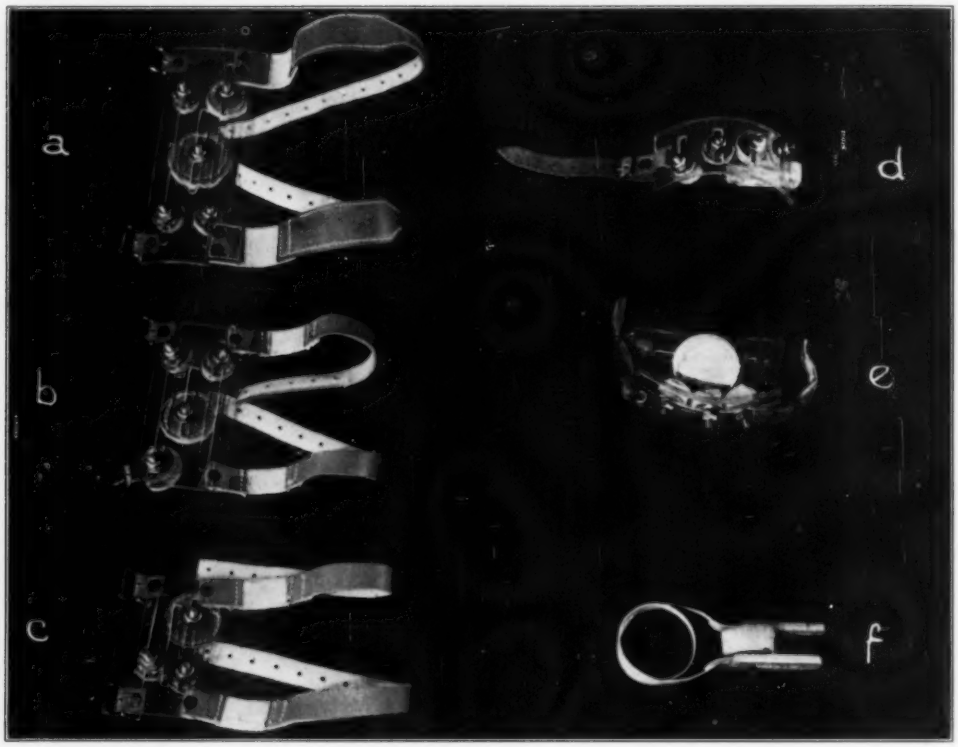

FIG. 2

Electrode holders with the electrodes in place: *a*. For sciatic, peroneal, and posterior tibial lesions. *b*. For median-nerve injuries, proximal muscle group. *c*. For ulnar nerve injuries, proximal muscle group. *d*. For radial-nerve injuries. *e*. For ulnar-nerve injuries, distal group. *f*. For median-nerve injuries, distal group.

This gives fairly accurate stimulation and, when consistently applied, the desired effect is obtained. This would indicate that, for practical purposes, the benefit arises from stimulation of the muscles to a maximum, whether separately or together; the stimulation of the antagonists should be avoided.

DESCRIPTION OF APPARATUS

The individual stimulator should be small, simple, inconspicuous, light, and with sufficient power to correspond to the stimulation previously applied. Through a series of models, one has finally been produced which meets most of these requirements. It consists of a small galvanic generator with a dry-cell battery (Fig. 1). Time and strength of the stimulus are controlled by adjustable switches. Intensity of the current may be regulated for arm or leg muscles. The current flow may be reversed by a polarity switch. The machine is shaped so that it may be carried in the trousers or tunic pocket with the electrodes applied to the desired area. The electrodes are of the usual construction, with two layers of one-quarter-inch white felt covering copper plates. They vary in size from five-eighths of an inch to one and one-half inches (Fig. 3). The electrodes are held in oblong plates of plexiglass, molded to fit the extremities (Fig. 2). These electrode holders have linear slots along which the electrodes may be moved to the points of maximum response. The holders are transparent to facilitate the accurate adjustment of the electrodes over motor points.

METHOD OF APPLICATION

The patient may be quickly and simply instructed in the application of the stimulator. He is taught to adjust the electrodes properly and to apply them over the paralyzed

muscles at the points of maximum stimulation. These points are marked for him by the physiotherapists. If he has difficulty in finding the proper areas, they may be marked by small brown tattoo points.

He is taught to place the extremity in the relaxed position (Fig. 4), to adjust the

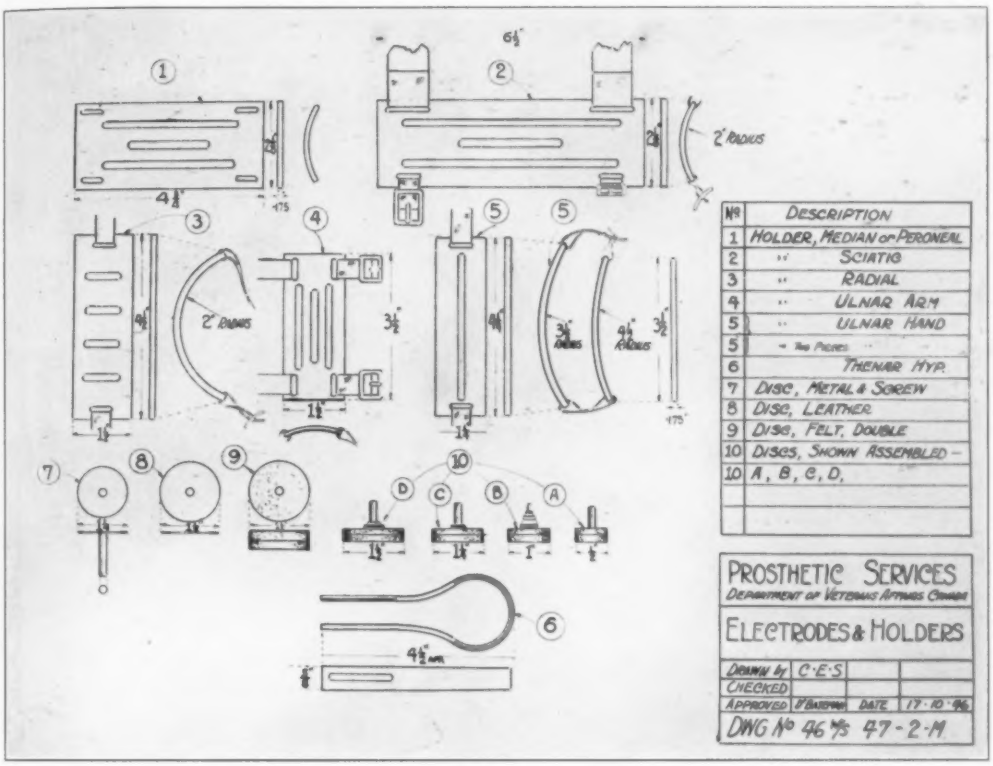

FIG. 3

Engineer's drawing, showing construction of the electrodes and the electrode holders.

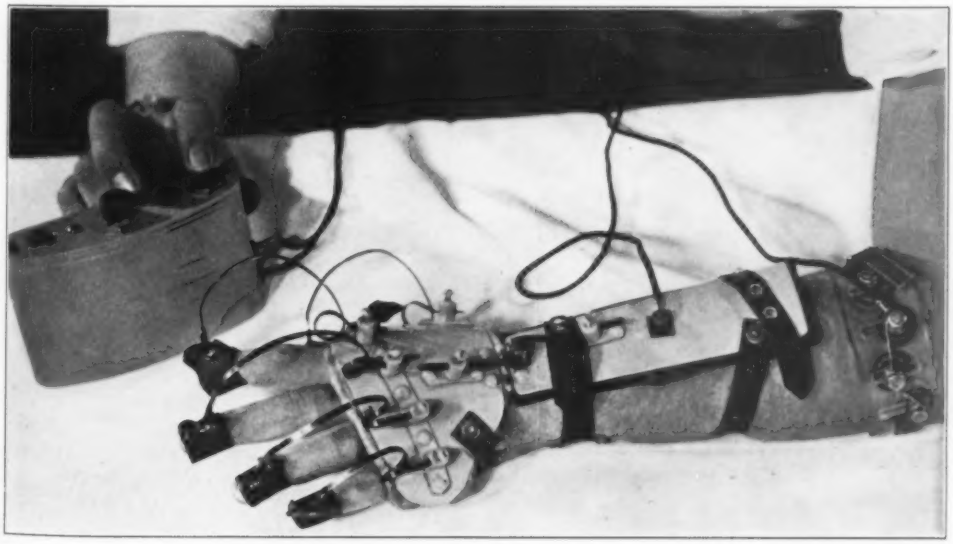

FIG. 4

The apparatus as applied to a hand splint in a case of radial-nerve paralysis. Note that the hand is placed in the relaxed position for treatment.

FIG. 5

Application of the electrodes for stimulation to the median proximal group of muscles.

FIG. 6

Showing method of stimulating median distal group.

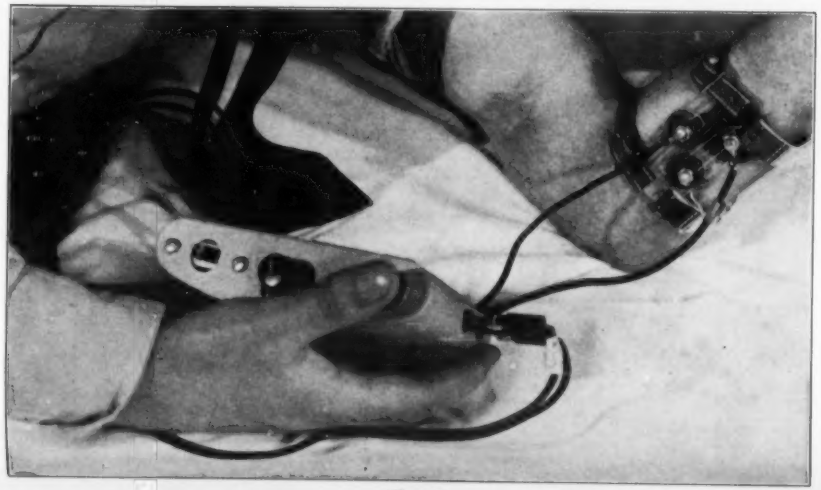

FIG. 7

FIG. 8

FIG. 9

Application for sciatic and posterior tibial groups.

FIG. 10

Stimulation of peroneal group through plaster.

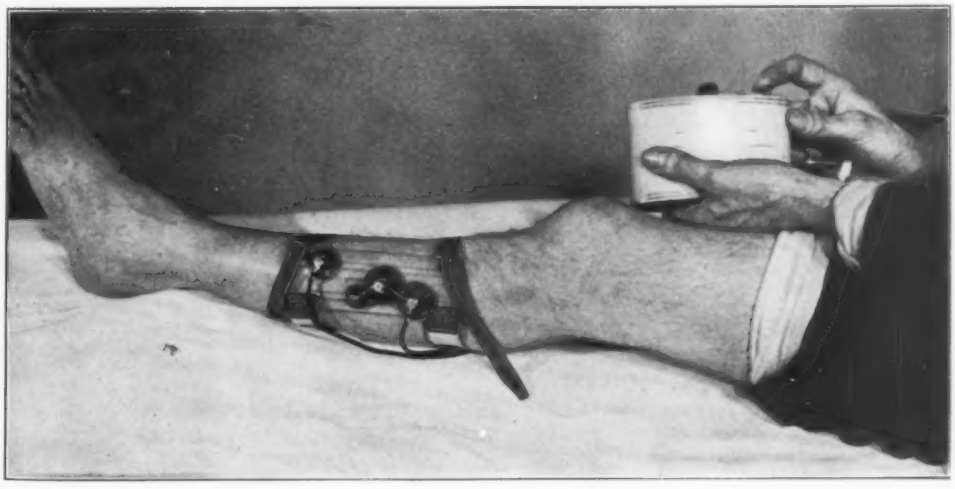

FIG. 11

Application for peroneal group out of plaster.

current until the desired response is obtained, and to avoid stimulating the antagonists. For maximum benefit, stimulation should be carried out hourly, producing ten to fifteen contractions of the paralyzed muscles. The stimulator may be easily carried in a pocket and the electrodes worn continuously, or, if the patient prefers, the electrodes may be carried and applied only for treatment. The electrodes are removed at night and allowed to soak in a weak saline solution. They are then ready for application the following day and will remain moist during the day.

RESULTS

1. The apparatus as outlined has been used on 150 patients and has functioned satisfactorily. A single battery will last five to seven months.

2. The apparatus is simple enough for the average patient to operate, and patients have displayed considerable intelligence in its application. Proper placing of the electrodes, the exact stimulus necessary, and the response that should be obtained are quickly learned.

3. The apparatus permits hourly treatment, instead of an irregular period every second or third day.

4. Patients may be discharged from the Hospital earlier, thus relieving the problem of accommodation. Readjustment and return to gainful occupation are facilitated.

5. The load of an overworked physiotherapy department has been lightened considerably.

CONCLUSIONS

The prospect of allowing the patient to treat himself was approached with hesitation, since carelessness, poor mentality, and lack of cooperation might spoil the whole plan. These fears have been groundless, because the more active participation under supervision has increased the patient's interest and cooperation. The danger of placing electrical therapeutic apparatus in the hands of the patient has been appreciated, and the unit has been constructed so that the patient cannot possibly do any real harm. The experiment has shown that proper electrical stimulation of muscles can be simply applied and continued for long periods by the average patient.

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AN IMPROVED RETRACTOR FOR INTERVERTEBRAL-DISC SURGERY

BY MICHAEL SKOVRON, M.D., ERIE, PENNSYLVANIA

Most surgeons who do intervertebral-disc surgery will agree that an adequate exposure is very important for the removal of a protruded intervertebral disc. This is accomplished by retracting the dura mater or the nerve root in the involved space. The author has used several types of retractors, but the one which proved most satisfactory is described herewith.

The retractor was made from a Simpson uterine sound, the rounded tip end of which was flattened thin (Fig. 1). The handle of the retractor is long and flat, thus preventing rotation. The shaft is made of soft metal which allows it to be bent in any shape for

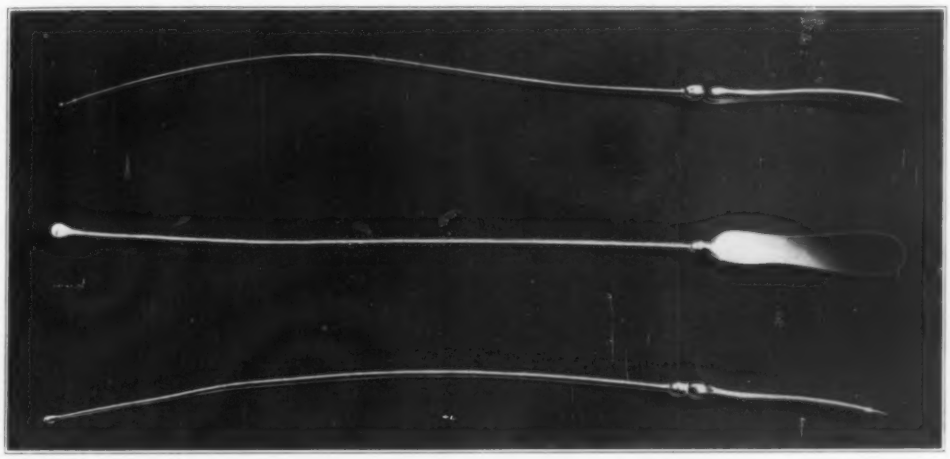

FIG. 1

A view of one retractor with a flattened tip after four years' use. The other two have not yet had their tips flattened, but show how the retractor may be bent,—convex below, and bayonet-type bend above.

retraction in different types of backs. The bayonet-type bend has been found to be most suitable.

The assistant holds the retractor in his left hand and a sucker in his right, using both sucker tip and retractor tip as blunt dissectors and retractors until the disc is exposed. Exposure is easily maintained with one hand, while the operator removes the protruded disc material. The assistant in no way interferes with the operator, because of the long shaft and handle of the retractor.

MODIFICATION OF THE DENIS BROWNE SPLINT

BY MICHAEL BLUHM, M.D., MEMPHIS, TENNESSEE

From the Campbell Clinic, Memphis

The most serious objection to the use of the Denis Browne splint in the treatment of congenital talipes equinovarus has been the difficulty of obtaining correction of the persistent adduction of the fore part of the foot[1]. It has long been recognized that this component of the deformity must be completely corrected before any attempt is made to correct the equinus.

Scarpa, as quoted by Adams, described a short leg brace with straps, which was used for maintaining position after operative correction of the club-foot deformity. Adams modified this by dividing the foot plate transversely, so that the mechanical center of motion corresponded with the mid-tarsal joint of the foot, this being the anatomical center of motion of the fore part of the foot with adduction deformity.

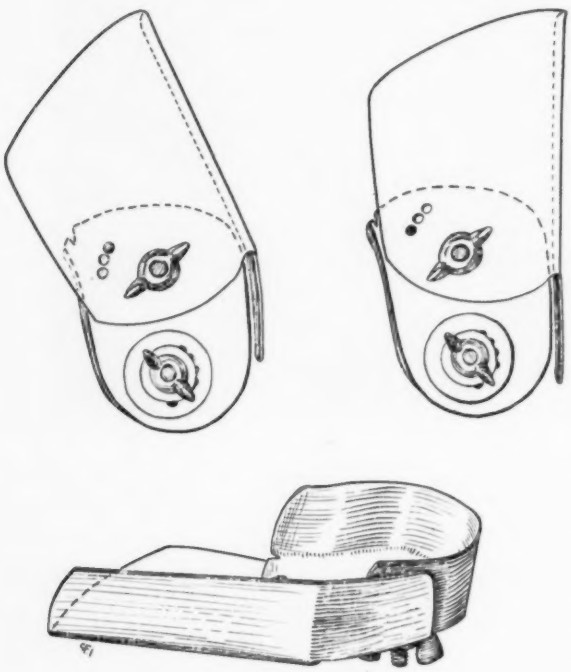

FIG. 1

This principle has been applied by the author to van Domselaar's modified foot plate of the Denis Browne splint, and it has been of definite value in obtaining correction of the adduction deformity of the fore part of the foot.

The foot plate (Fig. 1) is constructed in two parts,—a heel plate and a toe plate. The toe plate rotates freely in a transverse plane on a threaded axis pin of the heel plate. The pin is placed so that the axis of rotation corresponds to the center of the mid-tarsal joint. The degree of rotation is controlled by a series of holes in the toe plate and by a second pin on the heel plate. The flange on the inner side of the toe plate is extended backward, so that no defect will be present on the medial aspect of the foot plate which might allow window oedema to occur. This flange is malleable, and can be curved to conform to the shape of the medial aspect of the foot as the plate is gradually rotated externally. The sole of the splint is lined with white felt, and the flanges are lined with sponge rubber.

Farill has recently described a similar modification, using a worm screw to control the external rotation of the splint.

The treatment of talipes equinovarus by this modified plate is carried out in a manner similar to that outlined by Thomson, with the exception that the varus of the fore part of the foot is gradually corrected by the simultaneous external rotation of the toe plate, as well as by the external rotation of the entire plate itself on the cross bar.

NOTE: Mr. W. H. Broughton, foreman of the brace shop at Campbell Clinic, assisted in planning this foot plate, and also constructed it.

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MODIFICATION OF CAST SPREADER

BY FRED A. POLESKY, M.D., LOS ANGELES, CALIFORNIA

From the Department of Orthopaedic Surgery, College of Medical Evangelists and Cedars of Lebanon Hospital, Los Angeles

The cast spreader designed by the author is a modification of some of the cast spreaders which have been in use for many years. One advantage is that the spread of the cast is four and three-eighth inches (Fig. 1-A), which is a greater spread than can be obtained with most instruments now in use. In addition, the handles are so designed that, when the

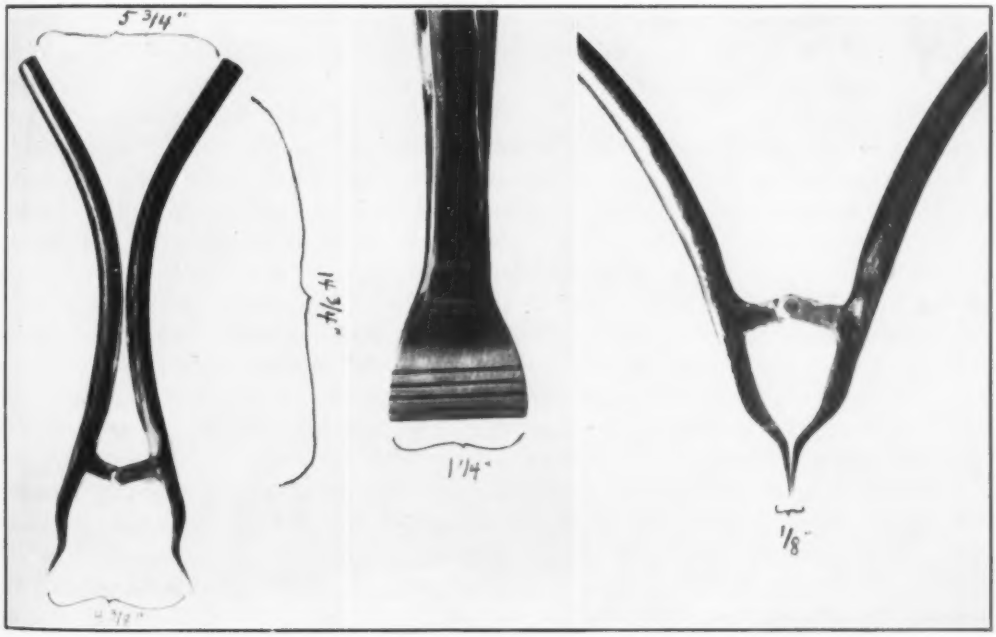

Fig. 1-A Fig. 1-B Fig. 1-C

cast is spread to the maximum, the handles do not come together. This means that the hands of the operator are not bruised at any time.

Another advantage is the length of the spreader, which is fourteen and three-quarters inches. This allows for a greater leverage, and consequently less effort is used in spreading a cast. The serrations on the outer side of the blade are necessary to prevent slipping of the instrument from the cast crevice after the cast has been split.

The cast spreader is made of light metal, and has been found very useful.

A REVIEW OF THE EVOLUTION OF THE ORTHOPAEDIC BRANCH OF SURGERY IN NEW YORK CITY

BY ROYAL WHITMAN, M.D., NEW YORK, N. Y.*

This review of the evolution of orthopaedic surgery properly begins with its christening by Valentine Mott in 1841.

He defined it as the "combination of operative with mechanical surgery, which had inaugurated a new and illustrious era in the healing art". This forecast has been amply justified; but, since the surgery of the combination was restricted to the subcutaneous division of contracted tissues in the correction of deformity, the "meridian splendor of its development", which he acclaimed in his own day, was deferred for many years,—until surgery in a comprehensive sense had become the dominant factor in its title, and the operative reconstruction of disabled members its distinctive feature.

Of those directly involved in Mott's pronouncement, Sayre, the only surgeon of the group, has been aptly termed its forerunner, since, as the first Professor of Orthopaedic Surgery, he included fractures, dislocations, and clinical surgery in his official title.

Knight, Davis, and Taylor, the upholders of the established practice, had each contributed a variant on the treatment of tuberculous disease of the hip and spine, then by far the most important constituents of orthopaedic practice. Thus they were at odds with Sayre in principle, and with each other in practice.

This was a period when, according to Holmes, "the atmosphere was vocal with the flippant loquacity of half-knowledge",—a characterization amply illustrated by the pioneers.

Sayre claimed that his subperiosteal resection of a tuberculous hip joint permitted reproduction of the bony structure, and thus the restoration of motion and stability in locomotion.

Davis claimed that his method of constant elastic extension actually separated the articulating surface of the hip joint, and thus, by permitting "motion without friction" during the period of repair, conserved the function of the joint and the nutrition of the limb.

Taylor claimed that his "jointed spinal assistant" checked flexion and pressure at the seat of disease; yet, by permitting voluntary extension of the spine, it utilized muscle action to restrain and to reduce deformity.

Knight rejected these "adventurous" expedients of his colleagues in favor of surgicomechanics and "expectancy", supplemented by enforced bodily activity to prevent the "etiolation consequent upon continued repose". He claimed that under this system 75 per cent. of the "ordinary conditioned patients laboring under synovitic disease were restored to self-sustaining ability".

It may be noted that these divergent methods were based on the mutually accepted principle that persistence in motion of a diseased joint, voluntary or enforced, was the essential preventive of ankylosis. Thus the local controversialists were united in their opposition to Thomas, the exponent of prolonged, uninterrupted rest as the basis of natural repair and, therefore, of conservation of function.

In 1887 the contentious pioneers and their associates, thirty-four in number, pooled their issues in organizing The American Orthopaedic Association. This event, from the evolutionary standpoint, was of almost equal importance to the christening, since it involved the question of identity. Whether its qualification as a specialty should be the means employed in treatment, or whether, as the representative of a new era, it was to be free from the restrictions associated with its ancient name,—this vital decision entailed an internecine conflict that lasted throughout the century.

* This article was submitted to *The Journal* by Dr. Whitman a few days before his death in August 1946. *Editor.*

The outstanding partisan of "mechanico-therapy" as the distinctive feature of the specialty was Newton M. Shaffer, who was a direct and consistent product of his early environment. He had served as a student assistant of Knight's in his primitive hospital; and, soon after his graduation in medicine, he had joined Taylor at the Orthopaedic Dispensary.

Knight's system of surgicomechanics included every disability amenable to relief by bandaging and bracing.

Taylor's range was by comparison far more limited. In later life, he stated that he had never written a prescription nor attempted a surgical operation, and that his formal medical education was completed in a single year. His original interest was in physical culture. For a time he conducted a gymnasium and devised many machines for automatic exercise. An unfortunate experience in the treatment of Pott's disease led to the invention of the "spinal assistant". Its conception was so novel and startling that, as he told it: "I jumped as if I had received a physical blow, and ran all the way home". By this inspired illumination, as it seemed to him, he became a convert to "mechanico-therapy" and eventually its leading exponent.

Shaffer, his disciple and successor at the New York Orthopaedic Hospital, claimed that its range had never been properly exploited, and that a rich reward awaited those who like himself were content to pursue these "underground but productive investigations". From this standpoint, the combination with operative surgery was impracticable, because one with this radical and superficially effective means at his command would be deflected from the prolonged, difficult, but far more adequate treatment by mechanico-therapy.

Shaffer was an energetic propagandist and, as such, he was particularly concerned with the recognition of the specialty, of which he was the sponsor, as on a par with those already established. He was a leader in the organization of The American Orthopaedic Association and in securing its admission to The Congress of American Physicians and Surgeons in 1888. On this occasion, he was its President and was elected as its delegate to the Council of the Congress. He was also chiefly responsible for the allotment of a special section to Orthopaedic Surgery at the International Congress held at Vienna in 1890.

At this Congress he presented his definition of the scope of the specialty which he represented,—namely, that it should be restricted to the class of cases for which especially designed mechanical appliances were required in treatment. Within these limits, operative intervention might be utilized in a subsidiary capacity, a concession of which he did not avail himself, and for which the Hospital under his control made no provision,—"For in true Orthopaedic Surgery, operative work *per se* has no real status".

Shaffer, as a Professor of Orthopaedic Surgery in a leading medical school and as surgeon-in-charge of the New York Orthopaedic Hospital, spoke as one in authority. As such, he exercised an arbitrary control of his assistants, permitting no divergence from his teaching and practice. Knight, under whom he had served his apprenticeship, had dismissed an able assistant of long standing, because in a publication on hip disease he had endorsed the traction treatment, "which weakened the vital resistance of the limb".

Shaffer, on the contrary, claimed that this method had "revolutionized the treatment of bone and joint disease". He was constrained, therefore, to contrive the dismissal of a former associate from his post at St. Luke's Hospital, because in a ward established by Taylor he had applied Thomas braces in the treatment of hip disease. This arbitrary extension of his authority brought to light the latent opposition to his personality and propaganda and, at the next meeting of The Association, he was displaced as its delegate to the Council of the Congress.

This episode has been considered at length, because the formal rejection of the limitations of scope and method, which Shaffer would have imposed, cleared the ground for a sustained advance, on which its establishment as a restricted specialty served only as a way station.

The leader of the progressive party during this preliminary period was Virgil P. Gibney, who was born and bred in the genial atmosphere of Kentucky. He completed his medical course at Bellevue, where he listened to the lectures of Sayre, whom he termed "that giant of Orthopaedic Surgery". Yet, immediately upon his graduation in 1871, he joined the staff of the Hospital for the Ruptured and Crippled, and remained as Knight's resident assistant for thirteen years.

On his dismissal, he took that opportunity "to brush up a bit" by visiting clinics in England and Scotland. He was particularly impressed by Thomas; although, as a convert to the traction method, he could hardly accept the conclusion that "it involved a fractional degree of fixation, sufficient to mask the evil of a ridiculous malpractice". On his return to New York, he began a private practice, in which he was immediately successful.

He joined Shaffer in the organization of The American Orthopaedic Association and was elected Chairman at its first session. In his representative capacity, he disclaimed "any intention to deny to any of his fellow members a special predilection for apparatus alone or for apparatus combined with minor operations or for operations alone". On the contrary, he invited all factions to unite in the establishment of a solidarity essential to the recognition of the nascent specialty.

In the same year, upon Knight's death, Gibney was appointed his successor and began at once what was to be his most important contribution to consistent progress,—namely, the transformation of a home for crippled children into a hospital, prepared in its professional and physical equipment to demonstrate the normal development of the orthopaedic branch of surgery. For this life-long task, Gibney had unusual qualifications. His long association with the Hospital assured the cooperation of the trustees in meeting the constantly increasing expense that the changes involved. He had, furthermore, the great advantage of building on Knight's foundation of surgicomechanics; free, therefore, from the artificial restrictions of departmental clinics and the physical limitation of hospitals for children.

Thus Knight, unwittingly, and Gibney, by intention, qualify as purveyors of evolution. In Knight's day, the two departments of the Hospital, although served by the same Staff, were practically distinct from one another. The majority of the indoor patients were "laboring under synovitic disease" and as the purpose of treatment was "to restore them to self-sustaining ability" the yearly admissions averaged less than 200. The out-patient department was far more important, since it carried out the main purpose for which the Hospital was incorporated,—namely, "to supply trusses, spring supports, bandages, laced stockings, and other suitable apparatus for the relief and cure of cripples, both adults and children, and to make these benefits available to the poorest in the community".

Gibney's first innovation on the established order was to improvise an operating room, although, to quote from the history of the Hospital, "at the age of forty he had had no experience in operative technique". This may have been an advantage in influencing his appointments to the Staff,—notably, that of Bull, a leading surgeon who was placed in charge of the Hernia Department.

As a recent recruit to surgery, Gibney, free from hampering preconceptions and personal prejudice, encouraged enterprise in his assistants and provided opportunity for its development. At this time, orthopaedic operations were, as in Mott's day, chiefly of the subcutaneous type and purely corrective in scope.

In 1929, when the physical development of the Hospital was fairly complete, 3522 operations were recorded. Of these, 1875 were classed as orthopaedic, and 1647 were credited to the Hernia Service. Thus, in the interval, the Hospital had become the leading exponent of constructive surgery, both for the ruptured and the crippled, confirming after many years Sayre's title of forerunner of a new era. Furthermore, under the impelling force of operative surgery, the Service had undergone radical reconstruction, in which its constituent factors had been expanded and coordinated to represent a progressive branch

of surgery. As such, the obsolescent, but indispensable, term "orthopaedic" designates its sphere of action,—namely, that of anatomical mechanics, of the correction of impairment of bodily structure and function from any cause, and of prevention as well as relief and cure.

Although it may be assumed that this conclusion, based on personal experience, is now generally accepted, a first-hand account of the background and the successive phases of this transformation, as epitomized in a hospital service, may be of interest to the present generation of orthopaedic surgeons.

" 'Tis fifty years since" when, as President of The American Orthopaedic Association, I presented conclusions on its scope and standing, which time has confirmed. Thus, as a participant, I have had an unusual opportunity to observe the formative stage of its development, with which this paper is especially concerned. Since the narrative is based upon personal experience, the field of action has been correspondingly restricted.

ENRIQUE H. LAGOMARSINO
1900–1946

News of the death of Enrique H. Lagomarsino on July 27, 1946, has recently been received. Dr. Lagomarsino was born in Buenos Aires, February 24, 1900. He received his medical degree in 1926, at the School of Medicine of the University of Buenos Aires, and then travelled and studied in Europe for several years. As part of his postgraduate work, he was an Assistant at the *Istituto Rizzoli*, in 1932.

Upon his return to Argentina, he became instructor and later Professor of Topographic Anatomy at the *Facultad de Ciencias Médicas de Buenos Aires*. He was also Professor of Orthopaedic and Traumatic Surgery of the School of Medicine of The University of La Plata, and was Chief of the Orthopaedic and Traumatologic Service at the Central Military Hospital.

Dr. Lagomarsino was a member of a number of societies, including The American Academy of Orthopaedic Surgeons, the Sociedad Argentina de Cirugía Ortopédica, the Sociedad Brasileira de Orthopedia e Traumatologia, and the Société Internationale de Chirurgie Orthopédique et de Traumatologie. He also held memberships in other surgical societies, in Bolivia, Chile, Paraguay, and Peru.

Among his several books, the best known are "Tratamiento de las Fracturas del Cuello del Fémur", written in collaboration with Dr. José Valls, and "Fracturas de la Columna vertebral". He made many contributions to Revista de Ortopedia y Traumatología and other surgical journals.

In a short span of forty-six years, Dr. Lagomarsino accomplished much in his chosen profession that will serve as a lasting memorial to him. He will be missed by his many friends and associates.

News Notes

The deadline for receipt of applications for Part I of the examinations of **The American Board of Orthopaedic Surgery** has been established as February 1, 1947.

Correspondence and applications should be forwarded to Dr. Francis M. McKeever, Secretary, 1136 West 6th Street, Los Angeles 14, California.

The National Society for Crippled Children and Adults, Inc., will hold its usual sale of Easter Seals. The purchase of these seals will help this important work. They may be obtained in quantities from The National Society for Crippled Children and Adults, Inc., 11 South La Salle Street, Chicago, 3, Illinois.

Announcement has been made of the formation of the **Sociedad Peruana de Ortopedia y Traumatología,** with headquarters in Lima, Peru. The purpose of the society is to encourage the study of the affections and traumata of the locomotor apparatus in all their aspects,—clinical, biological, surgical, and social. The President of the society for the current year is Dr. Oscar Guzmán, of Lima.

On October 12, 1946, the **New Jersey Orthopaedic Society** was organized at a meeting in Long Branch, New Jersey, with nineteen founder members, each of whom is a Diplomate of the American Board of Orthopaedic Surgery.

The following were elected to office:

President: Leopold Szerlip, M.D., Newark
Vice-President: Harold W. Smith, M.D., Orange
Secretary-Treasurer: Raphael R. Goldenberg, M.D., Paterson.

The National Foundation for Infantile Paralysis, Inc., announces the publication of a series of muscle charts. Under the direction of the committee on the treatment and after-effects of poliomyelitis, charts have been prepared giving a standard type of record of muscle strengths. These can be used advantageously in the care of poliomyelitic patients by the physical therapy departments of hospitals and clinics. They are available, free of charge and in quantities, and may be obtained from The National Foundation for Infantile Paralysis, Inc., 120 Broadway, New York 5, N. Y.

INTERNATIONAL SOCIETY OF ORTHOPAEDIC SURGERY AND TRAUMATOLOGY

The meeting of the *Société Internationale de Chirurgie Orthopédique et de Traumatologie* was held in Brussels, October 2, 3, 4, and 5, 1946, under the presidency of Dr. L. Ombredanne, of Paris. An interesting program was presented, with speakers from many different countries. Officers for the coming year were elected, as follows:

President: L. Ombredanne
Vice-Presidents: R. San Ricart and Harry Platt
Secretary-General: J. Delchef
Treasurer: C. Parisel.

Dr. Henry W. Meyerding, of Rochester, Minnesota, who attended the meeting as the delegate from the United States, was elected President of the next Congress, which will be held in Amsterdam, Holland, in the fall of 1948.

While Dr. Meyerding was abroad, he gave lectures at the Universities of Leiden, Amsterdam, and Edinburgh. He addressed the Society for Furtherance of Physics, Medicine and Surgery, in Amsterdam and the Société Française d'Orthopédie, in Paris. He also attended the 49th Congrès Française de Chirurgie, where he was elected to honorary membership in the Académie de Chirurgie Française which held its hundredth anniversary meeting in 1946.

THE BRITISH ORTHOPAEDIC ASSOCIATION

The Annual Meeting of the British Orthopaedic Association was held in London, October 18 and 19, 1946.

Mr. George Perkins, President of the Association, opened the meeting with a discussion on "Fractures of the Os Calcis". Mr. Norman W. Roberts and Mr. W. Sayle Creer had each followed up scores of cases over several years and had independently reached similar conclusions concerning compression

fractures involving the subastragaloid (subtalar) joint,—namely, that an incapacity period of about a year in cases treated by reduction and immobilization was approximately halved in cases treated by early movement. More than three-quarters of the patients returned ultimately to full work in their old occupations, and the proportion was not materially influenced by the method of treatment adopted. Mr. W. Gissane, nevertheless, considered reduction important, and showed a film illustrating its performance and maintenance with a sagittal os calcis pin. Mr. K. H. Pridie demonstrated some patients, including a policeman, who showed extremely good function following excision of the os calcis, an operation to be reserved for the most severely affected.

In his presidential address on Rest *versus* Activity in the Treatment of a Fracture, Mr. Perkins confined his remarks to fractures not directly involving joints. In stressing the importance of activity in preserving the extensibility of muscle, and, therefore, preventing stiffness, he pointed out that, if both fragments of a fracture bore muscles, they would afford immobilization, and the sole function of the limited splinting necessary was the preservation of alignment. In other cases (as in fractures of the femoral neck, the medial malleolus, and the scaphoid), splinting was necessary, not merely for alignment but also for immobilization. Such splinting must be prolonged, uninterrupted, rigid, and extensive; consequently, it impaired function, so that internal fixation was often preferable.

Professor E. Sorrel, of Paris, presented a paper on "*Les greffes d'immobilisation dans le traitement des tuberculoses osteo-articulaires*", in which the results of some usual and unusual extra-articular arthrodeses were illustrated. He strongly advocated arthrodesis as a final stage of treatment, to be deferred until active disease was at an end.

Professor H. J. Seddon showed a motion picture of preliminary work, carried out with Dr. A. E. Barclay, in the cineradiography of joint movements.

Mr. K. I. Nissen gave a comprehensive and up-to-date account of Morton's metatarsalgia, with a clinical and pathological description of thirteen cases, followed up for three years or more, in which he had excised the fibromatous thickening (first described by L. O. Betts of Adelaide).

Mr. I. Lawson Dick discussed the later results of iliac-bone transplantation, which were confirmatory of expectations.

Mr. L. Gillis showed a film of his operation for nearthrosis of the humeral shaft in amputations near the elbow joint, which has been described in the *British Medical Journal*.

At a clinical meeting at St. Thomas's Hospital, Dr. J. H. Cyriax presented some observations on dural pain in lumbago and sciatica. Cases were shown of open correction of congenital club-foot (Mr. G. Perkins); osteosynthesis by Kuntschner nail (Mr. R. H. Young) and by other methods of internal fixation (Mr. F. A. Simmonds); tendon transplantation for irrecoverable radial (musculospiral) paralysis (Mr. F. C. Durbin); tendon suture (Mr. R. Furlong); and the results of fractures of the tibial plateau (Mr. M. G. Allen) and of crush fractures of the spine (Mr. J. F. Bourdillon).

The Annual Dinner of the Association was honored by the presence of the Prime Minister, the Minister of Health, and the Minister of National Insurance as guests and speakers.

At the General Meeting, Mr. S. A. S. Malkin was elected President for 1948–1949. Professor E. Sorrel, of Paris, was elected an Honorary Fellow; Professor F. E. Godoy-Moreira, of Saõ Paulo, Kamel Bey Hussein, of Cairo, and Dr. Sven Kiaer, of Copenhagen, were made Corresponding Members. Mr. F. G. Allan and Mr. E. A. Nicoll were elected to the Executive Committee.

The following new members were elected:

G. P. Arden, F.R.C.S., Gerrards Cross, Buckinghamshire
Hiren De, F.R.C.S.E., Worcester
H. W. Fitzgerald, F.R.C.S.E., F.R.A.C.S., Dunedin, New Zealand
W. H. Gervis, F.R.C.S., Tunbridge Wells, Kent
Major A. P. Gracie, R.A.M.C., Newton Abbot, Devonshire
K. F. Hulbert, F.R.C.S., London, N.20
A. J. Innes, F.R.C.S., Glasgow, Scotland
T. B. McMurray, F.R.C.S.E., Liverpool
Miss M. G. Murphy, F.R.C.S.E., Harold Wood, Essex
Dr. B. McCall Smith, O.B.E., Glasgow, Scotland
F. G. Ward, F.R.C.S., London, E.C. 1.

THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS

The Fourteenth Annual Convention of The American Academy of Orthopaedic Surgeons will be held at the Palmer House, Chicago, January 25, 26, 27, 28, 29, and 30, 1947, under the presidency of Dr. J. E. M. Thomson.

Registration will begin at nine o'clock, Saturday morning, January 25. Scientific and Technical Exhibits will be ready for inspection at that time. During the afternoon the Audio-Visual Education

Program will be presented under the chairmanship of Dr. Charles N. Pease. At seven o'clock Saturday evening the Instructional Course Dinner will be given, at which President Thomson will read a paper on "Orthopaedic Surgery behind the Iron Curtain". Sir Reginald Watson-Jones will present a paper on "Personal Experiences as Consultant for the Royal Army Air Force".

The Audio-Visual Education Program will continue on Sunday morning from nine o'clock until noon. The Instructional Courses begin at two o'clock Sunday afternoon and run until noon on Monday. The following Courses will be offered:

Course I. Bone Tumors

Introduction	Dr. Henry W. Meyerding
The Pathologic Aspects	Dr. D. B. Phemister, Dr. Mary Sherman
X-Ray Diagnosis	Dr. Edward L. Jenkinson
Clinical Diagnosis and Operative Treatment	Dr. Henry W. Meyerding

Course II. Wrist and Hand

Wrist and Hand	Dr. Sterling Bunnell

Course III. Physiology of Bone

Nutrition, Mineral Metabolism, and Vitamins in Relation to Bone	Dr. F. C. McLean
The Endocrines and Bone	Dr. Edward C. Reifenstein, Jr.
Calcification and Decalcification of Bone	Dr. W. D. Armstrong

Course IV. Poliomyelitis

Diagnosis and Early Care of Poliomyelitis	Dr. James L. Wilson
Conservative Treatment of Poliomyelitis	Dr. Edward L. Compere
Operative Treatment of Poliomyelitis	Dr. William T. Green

Course V. Neck Lesions

Anatomy and Physiology of the Neck	Dr. K. B. Corbin
Differential Diagnosis	Dr. Ralph K. Ghormley
Injuries of the Cervical Spine	Dr. Arthur Davis
Non-Traumatic Lesions of the Neck	Dr. Carl Badgley

Course VI. Low-Back Lesions

Anatomical Considerations	Dr. Halford Hallock
Roentgenography	Dr. Albert Ferguson
Low-Back Syndrome	Dr. Paul Williams
Operative Treatment	Dr. Halford Hallock

Course VII. Congenital Dislocation of the Hip

Congenital Dislocation of the Hip	Dr. Joseph Freiberg
Early Management of Congenital Dislocation of the Hip, from Infancy to 8 Years of Age	Dr. H. R. McCarroll
Management of Congenital Dislocation of the Hip in Older Children	Dr. Bruce Gill

Course VIII. The Elbow Joint

Orthopaedic Anatomy	Dr. Harold Boyd
Differential Diagnosis and Treatment of Elbow Lesions	Dr. T. L. Waring
Traumatic Lesions About the Elbow	Sir Reginald Watson-Jones

Course IX. The Shoulder

Orthopaedic Anatomy of the Shoulder	Dr. John Fahey
Differential Diagnosis and Treatment of Common Lesions of the Shoulder	Dr. David M. Bosworth

Course X. Cerebral Palsy

Conservative Treatment of Cerebral Palsy	Dr. W. M. Phelps
Operative Treatment of Cerebral Palsy	Dr. Fremont A. Chandler

Course XI. Orthopaedic Anatomy of the Thigh

Orthopaedic Anatomy of the Thigh	Dr. J. E. Milgram

Course XII. Treatment of Congenital Club Feet

Treatment of Congenital Club Feet	Dr. Hiram Kite

Course XIII. X-Ray Diagnosis of the Extremities and Spine

X-Ray Diagnosis of the Extremities	Dr. Albert B. Ferguson
X-Ray Diagnosis of the Spine	Dr. Albert B. Ferguson

On Monday afternoon, Tuesday, Wednesday, and Thursday morning, the Scientific Program will be given, and Academy luncheons will be held at one o'clock on each of these days.

There will be Executive Sessions at noon on Tuesday and at noon on Thursday for members only.

The following program has been selected by the Program Committee under the chairmanship of Dr. Harold B. Boyd:

Monday, January 27

Afternoon Session

Treatment of Ununited Fractures by Onlay Grafts Without Screw or Tie Fixation.
Dallas B. Phemister, M.D.

Bone Grafts, an End-Result Study of the Healing Time.
W. A. Bishop, Jr., M.D.
Richard C. Stauffer, M.D.
Capt. Alvin L. Swenson, M.C.

A Method Combining a Bone Graft and Metal Plate in Non-Union of Long Bones.
G. Mosser Taylor, M.D.

Some Unusual Clinical and Pathological Aspects of Trauma.
C. Fred Ferciot, M.D.
Frank H. Tanner, M.D.

Preliminary Report from the Committee on Artificial Limbs.
Rufus H. Alldredge, M.D.

Tuesday, January 28

Morning Session

Slipping Epiphysis in the Adolescent Hip.
Paul H. Martin, M.D.

Malunited Displaced Upper Femoral Epiphysis. End-Result Study of Sixty-Five Cases Treated by Osteotomy of Surgical Neck with Internal Fixation.
Carl E. Badgley, M.D.
John Wolgamot, M.D.
James Miller, M.D.

Treatment of Congenital Dislocation of the Hip.
Professor Mudr. Bedřich Frejka, Brno, Czechoslovakia

Intertrochanteric Fractures of the Femur; A Survey of Treatment by Traction and Internal Fixation.
Mather Cleveland, M.D.
David M. Bosworth, M.D.
Frederick R. Thompson, M.D.

Principles of Treatment of Multiple Limb Injuries.
Sir Reginald Watson-Jones, London, England

Afternoon Session

Results of Recent Studies and Experiments Concerning Internal Fixation of Fractures with Metals.
Charles S. Venable, M.D.
Walter D. Stuck, M.D.

The Iliac Apophysis: An Invaluable Sign in the Management of Scoliosis.
Joseph C. Risser, M.D.

Anterior Tibial Tendon Transposition in Recurrent Congenital Club-Foot.
George J. Garceau, M.D.
K. R. Manning, M.D.

Arthroplasty of the Hip for Congenital Dislocation in Children.
Paul C. Colonna, M.D.

Coxa Plana.
M. B. Howorth, M.D.

WEDNESDAY, JANUARY 29

Morning Session

Growth of the Lower Extremity Affected by Anterior Poliomyelitis; Experiences in Equalizing Discrepancies by Epiphyseodesis.
William T. Green, M.D.
Margaret Anderson, M.S.

The Effect of Roentgen Irradiation on Epiphyseal Growth. Experimental Studies upon the Dog.
J. A. Reidy, M.D.
J. S. Barr, M.D.
J. R. Lingley, M.D.

Presidential Address.
J. E. M. Thomson, M.D.

Subtrochanteric Osteotomy in the Mobile Hip Controlled by the Well-Leg Traction Splint.
J. Warren White, M.D.
J. H. Turkell, M.D.

Our Experience in Treatment of Fracture of the Neck of the Femur.
José Luis Bado, M.D., Montevideo, Uruguay

Afternoon Session

Correction of Flexion Deformity in Spondylitis Deformans by Manipulation.
M. S. DeRoy, M.D.

Effect of Antireticular Cytotoxic Serum on the Healing of Fractures, A Preliminary Report of a Clinical Study.
Vernon Thompson, M.D.
Frederic W. Ilfeld, M.D.
John Grube, M.D.
Reuben Straus, M.D.
Moris Horwitz, M.D.

Veteran's Problem.
Paul B. Magnuson, M.D.

Styloidectomy of the Radius in the Surgical Treatment of Non-Union of the Carpal Scaphoid.
Leonard Barnard, M.D.
Sam G. Stubbins, Major, M.C.

The Contact Splint.
G. W. N. Eggers, M.D.

THURSDAY, JANUARY 30

Morning Session

Surgical Experiences with Injury to the Clavicle.
Thomas H. Peterson, M.D.

Fusion of the Hip in Children—Chandler Method.
Charles N. Pease, M.D.

Tuberculosis of the Hip in Children.
H. Relton McCarroll, M.D.
Robert D. Heath, M.D.

Flexor Tendon Grafts to the Fingers and Thumb.
Walter C. Graham, M.D.

Tendinous Reconstruction of the Hand Following Irreparable Peripheral Nerve and Brachial Plexus Injury.
C. A. Luckey, M.D.
S. R. McPherson, Capt., M.C.

The Annual Dinner will be held Wednesday evening, and on Tuesday evening a new feature will be added—an Historical Dinner. This will present the highlights of the lives of our earliest pioneers in orthopaedic surgery. The speakers are men who have had, in most instances, personal acquaintance with those whose lives they will review. Dr. Robert D. Schrock is Chairman of this project, and the speakers will be announced in the final program.